LOREN BROWN (Admitted *Pro Hac Vice*)
*loren.brown@us.dlapiper.com*
**DLA PIPER LLP (US)**
1251 Avenue of the Americas, 27th Floor
New York, NY 10020-1104
Tel: 212.335.4500 | Fax: 212.335.4501

MARY GATELY (Admitted *Pro Hac Vice*)
*mary.gately@us.dlapiper.com*
**DLA PIPER LLP (US)**
500 Eighth Street, NW
Washington, DC 20004
Tel: 202.799.4000 | Fax: 202.799.5000

BROOKE K. KIM SBN 239298
*brooke.kim@us.dlapiper.com*
**DLA PIPER LLP (US)**
4365 Executive Drive, Suite 1100
San Diego, California 92121-2133
Tel: 619.699.3439 | Fax: 619.699.2701

*Attorney for Defendant Nurture, LLC and Liaison Counsel for Defendants*

**UNITED STATES DISTRICT COURT**
**FOR THE NORTHERN DISTRICT OF CALIFORNIA**
**SAN FRANCISCO DIVISION**

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| IN RE: BABY FOOD PRODUCTS LIABILITY LITIGATION | Case No. 24-md-3101-JSC<br>MDL 3101<br><br>Hon. Jacqueline Scott Corley |
| This Document Relates to:<br>ALL ACTIONS | **DEFENDANTS' MOTION TO EXCLUDE PLAINTIFFS' CAUSATION/ EPIDEMIOLOGY EXPERTS (BRIEF 3)**<br><br>Date: December 8, 2025<br>Time: 9:00 a.m.<br>Location: Courtroom 8<br>19th Floor 450 Golden Gate Ave.<br>San Francisco, CA 94102 |

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## I. INTRODUCTION

At this general causation stage, Plaintiffs must come forward with reliable expert testimony opining that Defendants' baby food products are capable of causing Plaintiffs' alleged injuries—autism and ADHD. It is not enough for Plaintiffs' experts to argue that exposure to heavy metals via some other route, from some other source, at some other dose, or during some other time period of exposure can cause these conditions. *See* Ex. 53, at 38:14-16 (June 20, 2024, CMC Tr.).[1] Defendants make baby foods and/or sell them under their brands. These baby foods, some of which can contain trace levels of lead or arsenic, are the products Plaintiffs must show can cause the specific injuries they allege—autism and ADHD.

Plaintiffs' duty to meet their Rule 702 burden is particularly critical in this MDL because of the extraordinary nature of Plaintiffs' claim and its public health implications. First, there is no dispute that heavy metals are ubiquitous in our environment. As explained more fully in Defendants' Background brief, they are in our air, water, and soil (the earth is comprised of them), and thus become part of the food we eat. They cross the placental barrier and are found in breast milk. This means that all humans are exposed to heavy metals beginning at conception and continuing throughout early childhood and beyond—whether or not they eat commercial baby foods.

Second, even though lead in the environment has decreased by more than 90 percent in recent decades, the rates of autism and ADHD appear to have increased, and they are among the most common neurodevelopmental disorders in children today. Both disorders are primarily genetic in origin, and while there has been much scientific discussion and research about the *possible* role of non-genetic contributors, the few that have been identified occurred prenatally, and no recognized medical or scientific organization, regulatory authority, or pediatric, psychiatric, or neurologic society has ever concluded that consumption of heavy metals through food is a cause of these conditions. And as to the fruits, vegetables, or grains contained in baby foods, which can contain low levels of heavy metals, including lead and arsenic, there is no scientific evidence *at all* regarding

[1] Citations to "Ex." are to the exhibits attached to the Declaration of Livia M. Kiser, which is being filed simultaneously herewith.

any supposed causal role in autism or ADHD.

Third, Defendants' baby foods are known to be a complex mixture of many vitamins, nutrients, and other healthy constituents. The fruits, vegetables, and grains in baby food are universally accepted as critical to healthy infant development—and specifically to brain development—notwithstanding that some of them may contain trace amounts of lead or arsenic. In their work outside the courtroom, Plaintiffs' experts acknowledge the neurodevelopmental benefits of a nutritious diet.

Despite the lack of general (or really, any) acceptance of their causation theory, Plaintiffs proffer three epidemiologists (Drs. Ritz, Gardener, and Hu), two toxicologists (Drs. Aschner and Guilarte), and one clinical neurologist (Dr. Shapiro), all of whom claim—using virtually identical template language in their reports—that "lead *through* baby food" can cause some symptoms capable of being diagnosed as autism and ADHD and that "arsenic *through* baby food" can cause symptoms capable of being diagnosed as autism. When pressed, however, it became clear that none of these experts *actually considered food*. What Plaintiffs' experts offer, instead, is a syllogism: (1) lead and arsenic exposure can be neurotoxic in some settings and at some doses; (2) there is "no known safe level" of lead or arsenic; and therefore (3) *any dose* of lead or arsenic from *any source* via *any route of exposure* during *any early developmental window* could cause neurologic symptoms that may manifest as diagnosable autism and ADHD. Because some baby foods contain trace levels of lead or arsenic, the argument goes, baby foods themselves can cause symptoms diagnosable as autism/ADHD.

The methodologies that Plaintiffs' experts use to reach these opinions are unreliable and cannot satisfy Rule 702. Unable to rely on any scientific evidence relating to baby food that would reliably support their opinions, Plaintiffs' experts instead rest their opinions primarily on a body of epidemiologic studies that have investigated whether exposure to heavy metals in different contexts is associated with various neurodevelopmental outcomes or symptoms. These studies do not speak to the general causation question here because they involve different routes of exposure, different developmental windows, different (or unknown) sources and doses of heavy metals, different health outcomes, and different populations. Plaintiffs' experts fail to identify any reliable or scientifically

sound justification to make such an unfounded extrapolation.

Setting aside the analytical gaps between the literature these experts cite and the causation opinions about baby food they hope to offer, the studies themselves cannot reliably establish causation on their own terms. Most are case-control or cross-sectional studies that evaluate lead and arsenic exposure *after* diagnosis of autism/ADHD or whatever neurological symptoms are being studied, and, therefore, cannot make the critical showing that any heavy metal exposure occurred *before* the participants developed the outcome. In epidemiological language, these studies fail to satisfy the non-negotiable requirement of "temporality"—that the putative cause must precede the effect. These studies also fail to reliably control for major confounding factors, including genetics, family history, socio-economic status, and nutritional deficiencies that can, in some circumstances, increase retention and absorption of heavy metals.

In short, Plaintiffs cannot satisfy their burden of showing that sound science supports the claim that each of Defendants' baby foods can cause autism or ADHD. Plaintiffs' experts have failed to use reliable principles and methods to derive their opinions and have failed to reliably apply their methods to the facts in this case. *See* Fed. R. Evid. 702(c), (d); *see also General Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997) (requiring that an expert's opinion be based on a sound foundation and not a speculative methodology riddled with "analytical gap[s]" filled with nothing more than the experts' say-so); *In re Zantac (Ranitidine) Prods. Liab. Litig.*, 644 F. Supp. 3d 1075, 1191-92 (S.D. Fla. 2022) (excluding experts because the complete "lack of independent scientific support" for their opinions casts "doubt on the reliability of [their] methodology").

## II. BACKGROUND ON SCIENTIFIC PRINCIPLES RELEVANT TO GENERAL CAUSATION

Causation in toxic tort cases "is typically discussed in terms of generic [general] and specific causation." *In re Hanford Nuclear Reservation. Litig.*, 292 F.3d 1124, 1133 (9th Cir. 2002). General causation addresses "whether the substance at issue had the capacity to cause the harm alleged" at a population level. *Id.*

"The field of epidemiology addresses the incidence, distribution and etiology (causation) of disease in human populations by comparing individuals exposed to a particular agent to unexposed

individuals to determine whether exposure increases the risk of disease." *In re Bextra & Celebrex Mktg. Sales Pracs. & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1172 (N.D. Cal. 2007). A properly designed epidemiological study should consider both the size of the risk (meaning how much it is above, or below, a "no effect" level) as well as the statistical confidence in the finding (the confidence interval). Simply put, epidemiology is the way experts determine general causation, and both sides agree that human epidemiology is required to reach conclusions about causation for autism and ADHD. *See, e.g.*, *Hardeman v. Monsanto Co.*, 997 F.3d 941, 963 (9th Cir. 2021) ("To be admissible testimony, the experts must have reliably based their general causation opinions on epidemiological evidence showing a connection between glyphosate and cancer[.]"); Ex. 29, at 160:8-161:15 (Hu MDL Tr.) (testifying that epidemiology provides "a critical part of the evidentiary basis for determining causation"); Ex. 35, at 66:10-15 (Guilarte MDL Tr.) (agreeing that "epidemiological human data are required to reach causal conclusions about an exposure and ASD or ADHD in humans").

In many cases, the Bradford Hill framework is used to assess whether a "clear-cut association" between an exposure and a health outcome seen in epidemiologic studies can be said to reflect a true causal relationship. *See* Ex. 78, at 295-96 (Bradford Hill, *The Environment and Disease: Association or Causation?* (1965)); *see also* Federal Judicial Center, Reference Manual on Scientific Evidence ("Ref. Manual"), 597-606 (3d ed. 2011), Reference Manual on Scientific Evidence, Third Edition (2011).[2] Critically, however, the Bradford Hill analysis is not employed at all unless and until a valid association is established. *See*, *e.g.*, *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prod. Liab. Litig*. (*No. II)*, 892 F.3d 624, 642 (4th Cir. 2018) (affirming the trial court's finding that epidemiologic analysis of causation "requires a statistician to find a

[2] Bradford Hill identifies nine factors warranting consideration. These are (1) the strength of the association; (2) the consistency with which an association is shown across studies; (3) whether the association is specific to the outcome studied; (4) temporality—i.e., whether the observed effect occurs after the exposure; (5) biological gradient, also known as a dose-response relationship; (6) biological plausibility; (7) coherence—meaning that the scientific data make sense as a whole; (8) experimental evidence including challenge and de-challenge evidence; and (9) whether the association is analogous to other observed associations.

statistically significant association at step one before moving on to apply the [Bradford Hill] factors at step two"); Ref. Manual at 598-99 ("We emphasize that these [Bradford Hill] guidelines are employed only *after* a study finds an association to determine whether that association reflects a true causal relationship.") (emphasis in original) (citations omitted). This means that, before one applies Bradford Hill or any other metric for evaluating causality, an expert must assess whether the potential association is real, as opposed to the result of chance, confounding, or bias. *In re Mirena Ius Levonorgestrel-Related Prods. Liab. Litig. (No. II)*, 341 F. Supp. 3d 213, 265 (S.D.N.Y. 2018) ("[A]bsent such an association, there is no basis to apply the Bradford Hill criteria."), *aff'd sub nom. In re Mirena IUS Levonorgestrel-Related Prod. Liab. Litig. (No. II)*, 982 F.3d 113 (2d Cir. 2020).

Certain basic scientific principles apply to such assessments, as Plaintiffs' experts concede. The first is that "[a] temporal, or chronological, relationship must exist for causation to exist. If an exposure causes disease, the exposure must occur before the disease develops. If the exposure occurs after the disease develops, it cannot have caused the disease." Ref. Manual at 601. Cross-sectional studies, which examine exposure at the same time as the outcome of interest, or case-control studies that cannot establish that exposure preceded the outcome, by their very nature cannot demonstrate causation. Ref. Manual at 716 ("[I]n case-control and cross-sectional studies, the sequence of the exposure and outcome is unknown."). At the same time, even if a particular study *does* satisfy temporality, that does not prove causality: the fact that an outcome followed an exposure does not prove the exposure is causal.

Another foundational principle in causal assessments is that the "dose makes the poison." Ref. Manual at 637 ("The science of toxicology attempts to determine ***at what doses*** foreign agents produce their effects.") (emphasis added); Ex. 35, at 84:23-25 (Guilarte MDL Tr.) ("Q. Virtually any exposure can cause harm at a high enough dose, correct? A. Correct.") Thus, studies showing an association at a high dose cannot be used to extrapolate to a cause-and-effect relationship at a lower or much lower dose. Ex. 34, 67:2-68:2 (Aschner MDL Tr.) ("One would have to look very carefully at all the studies that have been published at a very low dose to see whether the same symptoms occur at low dose versus a very high dose."); *Joiner,* 522 U.S. at 137 (affirming lower court's rejection of expert's reliance on high-dose animal studies to infer causation related to

plaintiff's low-dose exposure). None of Plaintiffs' experts disputes this bedrock tenet of toxicology and causation.

It is also critical *not* to overstate the importance of, or the weight to be assigned to, certain factors in a causal analysis. For example, evidence suggesting that it is "biologically plausible" that a particular exposure would lead to a particular outcome is important for generating scientific hypotheses, but as with temporality, evidence of biological plausibility cannot be used to buttress an otherwise scientifically unsupported causal link in humans. Ref. Manual at 604-05.[3] Indeed, courts are cautious in their consideration of biological plausibility, particularly in cases where the biological mechanism by which the outcome at issue occurs is not well understood (as is the case with autism and ADHD), or where the proposed mechanisms are very generic and could thus play a role in even normal biological processes and conditions (as is the case with Plaintiffs' experts' proposed mechanisms here). *See Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996) ("[T]he courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it."); *see also Perry v. Novartis Pharms. Corp.*, 564 F. Supp. 2d 452, 468 (E.D. Pa. 2008) ("[T]he non-existence of good data does not allow [an] expert witness to speculate or base their conclusions on inadequate supporting science.")

## III. ARGUMENT

Plaintiffs' experts' causation opinions do not pass muster under Rule 702 because, at bottom, they rest on a series of unsupported analytical leaps and improper extrapolations from a body of literature that cannot answer the general causation question here. The incurable threshold problem with these experts' analyses is that they can point to *no* studies showing any connection between the consumption of baby food, recommended to begin at about six months of age and continuing into toddlerhood, and the development of autism or ADHD. As a result, Plaintiffs' causation experts must resort to a series of drastic leaps premised solely on studies that (1) do not involve food, (2) do not involve lead or arsenic doses to which children are exposed to heavy metals through baby food,

[3] *See also*, Ex. 35, at 77:4-12 (Guilarte MDL Tr.) ("Q. An association that is observed in an animal study may or may not hold true in humans, correct? A: Yes. Q. Okay. The only way to find out whether it will hold true in humans is to look at human data, correct? A: Correct.").

and (3) do not involve the correct endpoint (diagnosed autism or ADHD), population (U.S. children), or developmental window (early postnatal life when children eat baby food). None of these extrapolations are reliable or scientifically supportable.

Equally important, no reliable scientific evidence supports a causal relationship between heavy metal exposure *generally* and autism or ADHD. In other words, setting aside the fact that Plaintiffs' causation experts cannot reliably extrapolate from lead and arsenic studies to questions about baby food, the general heavy metal studies on which they rely cannot on their own terms provide a reliable basis for their opinions, which are necessarily reduced to the unsupportable position that heavy metal exposure through *any* route, at *any* dose, from *any* source, in any population, or during *any* developmental window can cause autism and/or ADHD.

### A. Plaintiffs' Experts' Causation Opinions Are Unreliable Because No Science Supports an Association Between Baby Food and Autism or ADHD.

Plaintiffs' causation experts' opinions cannot survive scrutiny under Rule 702 and must be excluded for multiple reasons. **First**, no scientific studies show an association between baby food or any other food and an increased risk of autism or ADHD, nor is it generally accepted that eating healthy food causes autism or ADHD. As such, no methodologically reliable basis exists for Plaintiffs' experts to offer general causation opinions. **Second,** Plaintiffs' experts' causation opinions cannot be reconciled with the widely held scientific consensus that the kinds of fruits, vegetables, and grains found in Defendants' baby food products play a key role in healthy brain and overall development in infants and children. **Third**, Plaintiffs' experts simply ignore and/or refuse to engage with the scientific fact that food is a complex mixture containing dozens of micronutrients (*e.g.*, vitamins and minerals), macronutrients (*e.g.*, fats, proteins, carbohydrates), and other constituents (*e.g.*, fiber) that interact to affect the extent to which lead and arsenic are released from foods, absorbed through the gut, and transported to and retained (or not) in the body. These experts also ignore the science demonstrating that the nutrients and other constituents that make up food independently produce *beneficial* neurodevelopmental effects. **Fourth**, Plaintiffs' experts fail to bridge the analytical gap created by the absence of *any* food studies by pointing to studies of single nutrients. They claim that there is no reliable scientific evidence that certain single nutrients present

in baby foods affirmatively *prevent* the bodily uptake of lead and arsenic or the alleged neurotoxic effects of these metals, but that circular, burden-shifting maneuver neither accounts for the lack of a demonstrated effect of food in the first place, nor can it address the general causation question here.

**1. No Studies or Any Other Scientific Evidence Link Baby Food, or Food Like Defendants' Baby Food, to Autism or ADHD.**

Plaintiffs' causation experts' opinions all fail for the simple reason that Plaintiffs lack *any* scientific evidence regarding the actual general causation question here: whether Defendants' baby food products are capable of causing autism or ADHD. There are, in fact, no such studies. *See, e.g.*, Ex. 35, at 52:9-19 (Guilarte MDL Tr.) (testifying that he could not name "any peer-reviewed publication that states that there's a causal association between baby food or heavy metals in baby food and ASD or ADHD"); Ex. 34, at 112:7-8 (Aschner MDL Tr.) ("There's no studies on baby food and health effects in my report."); Ex. 29, at 303:13-25 (Hu MDL Tr.) (testifying that none of the literature that he considered referenced baby food specifically).

Nor do Plaintiffs' experts identify any scientific evidence linking consumption of foods rich in grains, vegetables, and fruits, which are generally accepted to have trace levels of heavy metals comparable to levels in homemade baby foods containing similar ingredients, to an increased risk of autism or ADHD. *See, e.g.*, Ex. 43, at 89:11-23 (Ritz Landon Tr.) (testifying that she evaluated studies that measured metal in blood, not food). Thus, although Plaintiffs' experts claim to be offering opinions about Defendants' baby food products,[4] they fail to identify any evidence relating to those products (or even as to the ingredients comprising those products). That complete lack of product-relevant data, coupled with the inability to draw reliable, product-relevant conclusions based on the studies on which these experts *do* rely, should end this Court's Rule 702 inquiry. *See Perry*, 564 F. Supp. 2d at 467-68 ("In cases where no adequate study shows the link between a substance and a disease, expert testimony will generally be inadmissible, even if there are hints in

[4] *But see* Ex. 35, at 25:18-20 (Guilarte MDL Tr.) ("I don't believe I said that baby food [causes autism or ADHD]; I said the toxic metals in baby food will do that.").

the data that some link might exist."). "[T]he courtroom is not the place for scientific guesswork." *Rosen*, 78 F.3d at 319.

**2. Plaintiffs' Experts' Theories Not Only Lack General Acceptance, But They Also Are Contrary to Established Science That Fruits, Grains, and Vegetables Are Healthy for Brain Development.**

Not surprisingly, given the absence of any supportive evidence, the opinions Plaintiffs' experts seek to offer find no general acceptance in the scientific community. To the contrary, the scientific community overwhelmingly recognizes that fruits, grains, and vegetables, which are found in commercial baby food, are critical to a healthy diet and brain development. General acceptance in the relevant scientific community remains a key factor in assessing the reliability of an expert's opinion under Rule 702. *Daubert v. Merrell Dow Pharmas., Inc.*, 509 U.S. 579, 594 (1993); *United States v. Halamek*, 5 F.4th 1081, 1087 (9th Cir. 2021). No major medical or scientific organization has suggested that commercial baby food causes autism and/or ADHD, or that heavy metals at the levels found in foods generally and commercial baby food specifically cause autism and/or ADHD. *See, e.g.*, Ex. 26, at 206:10-208:5 (Ritz MDL Tr.); Ex. 29, at 300:12-19 (Hu MDL Tr.) ("Q. Are you aware of any medical, scientific, or regulatory organization which has concluded that baby food can cause autism? . . . THE WITNESS: I'm not aware of any scientific organization that has even considered the question."). Just this week, the American Academy of Pediatrics reiterated that genetics (not food) is the primary driver of autism: "[r]egarding autism, we know it is complex, highly variable and increasingly linked to genetics." AAP Statement on White House Autism Announcement (Sept. 22, 2025), AAP Statement on White House Autism Announcement. Even Plaintiffs' own experts have never published a peer-reviewed study or other research linking commercial baby food to autism and/or ADHD. *See, e.g.*, Ex. 49, at 350:1-21, 402:11-17 (Shapiro MDL Tr., Vol. II).

What the scientific community overwhelmingly recognizes is that fruits, grains, and vegetables, like those found in commercial baby food, are not just critical for healthy brain development and overall wellness; they may even help prevent any harmful effects of heavy metal exposure. *See, e.g.*, EPA, Actions to Reduce Potential Lead Risk (2025), Actions to Reduce Potential Lead Exposure | US EPA ("Eat a well-balanced diet of fruits, vegetables, grains, dairy and protein-

rich foods. Foods that are higher in calcium, iron and vitamin C can help reduce the body's absorption of lead. ***Children with healthy diets absorb less lead.***") (emphasis added); FDA, Help Protect Children from Environmental Contaminants: Healthy Food Choices for Your Baby Aged 6-12 Months (2023), Help Protect Children from Environmental Contaminants: Healthy Food Choices for Your Baby Aged 6-12 Months | FDA ("Iron and zinc are essential nutrients for child development. They also can help prevent the harmful effects of arsenic, lead, cadmium, and mercury."); Cal. Dep't of Pub. Health, Well Fed = Less Lead (2021) ("Full bellies absorb less lead."). Even acknowledging the presence of heavy metals in commercial baby food, the FDA recommends that children continue to eat commercial baby food. *See* FDA, FDA Issues Final Guidance for Industry on Action Levels for Lead in Processed Food Intended for Babies and Young Children (2025), FDA Issues Final Guidance for Industry on Action Levels for Lead in Processed Food Intended for Babies and Young Children | FDA ("Parents and caregivers should not throw out processed or packaged baby foods or stop feeding certain foods to babies and young children.").

That Plaintiffs' experts' opinions are contrary to the widely held belief in the scientific community regarding the vital role baby food plays in child nutrition and brain development is a key indicator that these opinions are unreliable and must be excluded under Rule 702. *Daubert*, 509 U.S. at 594; *Ibok v. Advanced Micro Devices, Inc*., No. 5:02-cv-01485, 2003 U.S. Dist. LEXIS 27623, at *10-11 (N.D. Cal. July 2, 2003) (excluding expert analysis which "has not been subject to peer review and has not achieved general acceptance" and is therefore "unreliable and inadmissible").

**3. Plaintiffs' Experts Acknowledge That Food Is a Complex Mixture of Vitamins and Nutrients, but They Unreliably Treat Food Like Any Exposure Source.**

Plaintiffs' experts also cannot survive under Rule 702 because they ignore generally accepted science regarding the very nature of healthful food. Plaintiffs' experts (with one exception) do not dispute that food is a complex mixture containing a wide array of nutrients, vitamins, and

other constituents that are essential for both overall and neurological development.[5] But when it came to preparing their opinions in this case, these same experts failed to contend with this accepted science and how it might impact their analysis, instead treating food as if it were just an exposure source like air or water or even paint chips.

At deposition, Plaintiffs' experts repeatedly admitted that nutrients and other constituents in food interact and must be considered as a "whole package" when assessing health effects. *See, e.g.*, Ex. 43, at 54:19-55:6 (Ritz Landon Tr.). As Dr. Ritz explained, one must look at food as a complex mixture because "there is always an interaction" in food, such that the whole is different from the individual parts. *Id.* at 54:19-55:6. Dr. Hu also testified that "without looking at the effect of multiple nutrients, that is a limitation; you don't get the full picture." Ex. 29, at 187:1-12 (Hu MDL Tr.). Similarly, Dr. Aschner agreed that food "is a very complex mixture of nutrient and non-nutrient substances," and that just looking at "lead and iron … in isolation and forgetting about everything else does not depict the biological picture." Ex. 34, at 248:9-12 (Aschner Landon Tr.); *id*. at 280:2-5; *see also id.* at 292:21-294:12 (agreeing that studies looking at the "totality of everything in the diet" are necessary to assess the impact of food on pathways relevant to his analysis but stating that those studies are generally not available). Dr. Shapiro also did not dispute this point: "If your question is [] how food contributed to someone's neurodevelopment, you do have to consider all the components of that food." Ex. 36, at 353:25-354:14 (Shapiro Landon Tr., Vol. I).

Indeed, when Plaintiffs' experts have studied food outside of the courtroom as part of their academic work, they have treated food as the complete and complex mixture that it is, conceding

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[5] In contrast to Plaintiffs' other experts, Dr. Gardener testified in both the California litigation and here that, remarkably, she does not know whether food is a complex mixture. When asked whether she agreed that whole foods are "complex structures that include a variety of nutrients, vitamins, and phytochemicals," Dr. Gardener replied, "I'm not prepared to opine on whether that's a true statement or an untrue statement." Ex. 46, at 160:11-19 (Gardener Landon Tr.). More recently, when asked whether baby food is a complex mixture, she testified, "[s]o some baby foods are simple; they'll just be a single ingredient. And other baby foods have multiple ingredients. Some will have cinnamon, like I talked about earlier. A lot of the applesauce pouches have cinnamon. I don't know if you would call that a complex mixture." Ex. 33, at 289:3-9 (Gardener MDL Tr.). Dr. Gardener's unwillingness to acknowledge even a basic scientific principle when Plaintiffs' other experts admit that even individual foods, like apples, contain hundreds of chemical constituents, betrays any notion that she is acting as a scientist rather than an advocate for Plaintiffs.

that it may have beneficial effects on certain health outcomes. Dr. Ritz, for example, has found that a nutritious diet can have a favorable impact on the development of other neurological conditions such as Parkinson's disease. *See* Ex. 43, at 122:2-8 (Ritz Landon Tr.) (considering the impact of food on Parkinson's outcomes, Dr. Ritz stated "we did not look at supplements. We only looked at the diet."). Dr. Gardener has studied the neurological benefits of a Mediterranean diet, and Dr. Hu's research has addressed the potential harms of a Western diet. *See* Ex. 46, at 50:22-51:8, 149:22-7 (Gardener Landon Tr.); *see also* Ex. 29, at 184:6-20 (Hu MDL Tr.).

Dr. Ritz also has participated in research examining the interaction between contaminants that are capable of promoting oxidative stress and an antioxidant-rich diet. *See* Ex. 26, at 231:25-232:18, 234:14-236:25 (Ritz MDL Tr.). In a recently published study, Dr. Ritz and her team concluded that consuming "a diet high in vitamin C and other antioxidants *may potentially counteract oxidative stress* in the body." Ex. 28, at 1, 4 (Ritz MDL Tr., Ex. 18) (Yu, et al., *Diet, polycyclic aromatic hydrocarbons, and oxidative stress biomarkers in pregnancy: A Los Angeles pregnancy cohort* (2025)) (emphasis added) (stating also that "better adherence to a Mediterranean diet pattern and the dietary guidelines for Americans were associated with lower oxidative stress biomarker levels;" and "a diet rich in antioxidants may ameliorate oxidative stress by neutralizing reactive oxygen species[.]"). Oxidative stress is one of the potentially harmful effects that Dr. Ritz and other Plaintiffs' experts opine can result from exposure to heavy metals like lead and arsenic and contribute to autism and ADHD.

Consistent with this finding, Dr. Ritz admitted at deposition that nutrients in food can favorably impact the same biological processes that lead and arsenic allegedly impair:

> Q. Folate, B vitamins, choline and vitamin C can all impact DNA methylation in a favorable way. Isn't that true?
>
> A. That's correct.
>
> Q. A nutritious diet also has an impact on oxidative stress in the body. Is that right?
>
> A. Generally, yes.
>
> Q. A nutritious diet can interact with and neutralize what are known as free radicals. Is that right?
>
> A. As general as that, yes.
>
> …

Q. Okay. Is it true that a nutritious diet can also impact fetal growth in a favorable way?

A. Yes.

Q. And a nutritious diet can also reduce inflammation in a body. Is that right?

A. We believe in general that that's possible, yes.

Ex. 26, at 225:24-227:9 (Ritz MDL Tr.); *see also* Ex. 43, at 167:13-16 (Ritz Landon Tr.) (testifying that whole grains may be fortified with folic acid to ensure folic sufficiency).

Despite repeatedly acknowledging these (uncontroversial) principles, *none* of Plaintiffs' experts' opinions reliably account for the ways that exposure to lead and arsenic *in healthy foods containing essential nutrients* differs from exposure to the same heavy metals through exposure sources like water, air, or soil. For example, notwithstanding her admissions—before and during this litigation—that a healthy, nutrient-rich diet can counteract oxidative stress, Dr. Ritz did not even consider "whether the net effect of eating fruits and vegetables with trace amounts of heavy metals at the same levels found in baby food would result in a net reduction in oxidative stress in the body or a net increase in oxidative stress in the body." Ex. 26, at 238:13-239:4 (Ritz MDL Tr.); *see also* Ex. 46, at 162:9-163:3 (Gardener Landon Tr.) (stating "I'm not prepared to opine on—on those—those interactions," in response to whether she had considered if antioxidants in food offset any oxidative stress from heavy metals in the brain).[6]

Dr. Hu readily agreed that the nutrients he understands to be in baby foods "have a beneficial impact on brain health and neurodevelopment." As he put it: "Of course they do." Ex. 29, at 24:22-25:14 (Hu MDL Tr.). But Dr. Hu also admitted that he did not investigate the composition of Defendants' foods, review any of the relevant literature, or do anything else to determine whether the net impact of baby food on neurodevelopment is positive or negative, including with respect to

[6] Indeed, Dr. Ritz did virtually *nothing* to understand the composition of Defendants' baby foods or to use product-specific data in forming her expert views. She reviewed an Excel summary of certain label information provided by Plaintiffs' counsel that included vitamin C, iron, vitamin A, vitamin E, vitamin D, and folate information, "but that's basically it." Ex. 26, at 228:2-229:25 (Ritz MDL Tr.).

autism/ADHD diagnosis risk. *Id.* at 192:14-22 ("Q. Are there bodies of literature that evaluate the neurodevelopmental impacts of any particular vegetables, fruits, or grains? MS. FORGIE: Objection. THE WITNESS: I haven't looked at that literature from that specific view, but I would venture that those—those characteristics have been researched extensively."). Nor did Dr. Hu conduct any analysis of how any food containing both trace metals and nutrients may impact neurodevelopment or autism/ADHD, much less the net neurodevelopmental impact of *baby food* on a healthy brain. *See id.* at 191:17-192:3, 193:18-21 (failing to evaluate foods and impact on autism/ADHD). Dr. Hu's decision to ignore such important data is particularly striking considering that certain of the data are reflected in his own publications. *See, e.g.*, Ex. 32 (Hu MDL Tr., Ex. 13) (Wang, *et al*., *A Western Diet Pattern Is Associated with Higher Concentrations of Blood and Bone Lead among Middle-Aged and Elderly Men* (2017)); Ex. 29, at 207:11-209:7 (Hu MDL Tr.) (stating nutritious foods like berries and certain vegetables can have antioxidant effects); *id.* at 210:10-24 (agreeing with his prior publication statement that "Vitamin C, as an antioxidant, can scavenge lead-induced free radicals"). Dr. Hu has no explanation for why he failed to take those principles into account in reaching his causation opinion here.

Similarly, despite having concluded in her academic work that a diet rich in antioxidants and anti-inflammatory agents can have a protective effect on brain health in adults,[7] Dr. Gardener did nothing in this case to investigate whether there have been similar findings in children: "I have not reviewed that literature to be able to sit here and—and opine about that line of questioning." Ex. 46, at 149:15-150:13 (Gardener Landon Tr.) (emphasis added); *see also id.* at 162:9-163:3 (stating "I'm not prepared to opine on—on those—those interactions," in response to whether she had considered if antioxidants in food offset any oxidative stress from heavy metals in the brain) Remarkably, notwithstanding the fact that she is offering an opinion that heavy metals in baby foods can cause autism, Dr. Gardener disclaimed any obligation to consider the association between food and autism

[7] In a peer-reviewed article published earlier this year, Dr. Gardener wrote that "increased consumption of calcium and iron may act as antagonists [inhibitors] for lead and cadmium[.]" Ex. 84, at 4-5 (Gardener, *et al*. *Heavy metals and phthalate contamination in prenatal vitamins and folic acid supplements* (2025)).

as outside of her assigned task. In her words, "I am not here to opine on the association between vegetable consumption and autism risk, other than to the extent that vegetables are an important source of heavy metals and heavy metals are associated with—with autism risk." *Id.* at 104:14-23.

Dr. Aschner, Dr. Guilarte, and Dr. Shapiro took similarly irreconcilable positions. Despite acknowledging that healthy food is important and beneficial, none of these experts attempted to assess the potential interactive effect of healthy nutrients and heavy metals when consumed in food. *See, e.g.,* Ex. 34, at 123:2-16 (Aschner MDL Tr.) ("[E]ating healthy food is important. Everybody agrees."). Dr. Aschner claimed that because he is not a nutritionist, he could not opine on the role of antioxidants in food on children's development and said it was "beyond [his] charge" to assess the net effect of food on children's neurodevelopment. *Id.* at 123:22-124:6. Dr. Guilarte similarly testified that he had not "done a specific evaluation of whether each nutrient and vitamin in baby food can counteract the effects of lead and arsenic in baby food" or "whether all the nutrients in baby food together can counteract the effects of lead and arsenic in baby food." Ex. 35, at 325:3-17 (Guilarte MDL Tr.). When asked if he has "done an evaluation or research into whether whole food with all of its nutritional components, vitamins and nutrients causes the mechanistic theories and hypotheses that [he] postulate[s]," Dr. Guilarte answered: "I can't answer that question. I don't know . . . I don't think that study has been done." *Id.* at 364:6-19. And Dr. Shapiro testified that he had not even looked at the specific products in this litigation and could not speak to the vitamins, minerals, nutrients, and other components in those foods. Ex. 36, at 152:12-153:5 (Shapiro MDL Tr., Vol. I).

"Sound scientific methodology in assessing general causation requires an expert to evaluate 'all of the scientific evidence when making causation determinations.'" *Daniels-Feasel v. Forest Pharms., Inc.*, No. 17 CV 4188-LTS-JLC, 2021 WL 4037820, at *5 (S.D.N.Y. Sept. 3, 2021) (quoting *In re Zoloft (Sertraline Hydrocloride) Prod. Liab. Litig.*, 26 F. Supp. 3d 449, 463 (E.D. Pa. 2014)). Plaintiffs' experts' failure to account for, or even *consider*, evidence regarding the known benefits of the kinds of nutrients in Defendants' baby foods in reaching their causation opinions is the very definition of a result-oriented and unscientific methodology. And their contrary statements outside the courtroom—including in their own published research—make it abundantly clear that

these experts are not "employ[ing] the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152 (1999).

**4. Plaintiffs' Experts Ignore the Extensive Literature About Food, Which Demonstrates the Unreliability of Their Opinions.**

Given that their opinions are contrary to the scientific community's conclusion on the healthful impacts of food, it is unsurprising that Plaintiffs ignore the extensive body of literature involving food. Studies of nutrition, dietary patterns, and early neurodevelopmental outcomes *do* exist—they just do not support Plaintiffs' claim that baby food can cause autism or ADHD. Plaintiffs' experts omit from their reports (and some exclude from their materials considered) studies that examine whole food consumption, blood lead levels, and autism diagnosis, and find that children who consume more fruits, vegetables, and seafood[8] have *lower* rates of autism. *See, e.g.*, Ex. 27 (Ritz MDL Tr., Ex. 10) (Rahbar, et al., *Concentrations of Lead, Mercury, Arsenic, Cadmium, Manganese, and Aluminum in the Blood of Pakistani Children with and without Autism Spectrum Disorder and Their Associated Factors* (2021)); Ex. 26, at 105:2-13; 107:6-16 (Ritz MDL Tr.) (explaining Rahbar 2021 not a study on Ritz's reliance materials); *see also* Ex. 44 (Ritz Landon Tr., Ex. 16) (Rahbar, et al., *Detoxification Role of Metabolic Glutathione S-Transferase (GTS) Genes in Blood Lead Concentrations of Jamaican Children with and without Autism Spectrum Disorder* (2022)); Ex. 43, at 151:15-152:11, 157:3-9, 158:1-10 (Ritz Landon Tr.) (discussing findings of Rahbar, et al., (2022) that children with autism had lower blood lead levels and "significantly lower consumption of various types of fruits and vegetables as well as seafood that are potential dietary sources of lead exposure in children"); Ex. 34, at 288:8-14, 290:1-17 (Aschner MDL Tr.). In those studies, researchers found that children *without autism* ate significantly *more* yogurt, apples, strawberries, and root vegetables such as carrots and sweet potatoes than children with autism.[9] *See*

[8] Seafood and fish are not found in Defendants' baby foods.

[9] Some of these studies assessing food do not study a U.S. population, which presents challenges with generalizability. But, as discussed, *infra*, Plaintiffs' experts rely heavily on lead and arsenic studies that do not assess U.S. populations and yet cannot explain why they fail to consider ex-U.S. *food* studies. This is classic results-driven cherry-picking that renders an expert opinion

Ex. 26, at 108:3-111:3 (Ritz MDL Tr.); *see also* Ex. 74, at 8 (Table 2), 9 (Table 3) (Rahbar, M., et al., *Concentrations of Lead, Mercury, Arsenic, Cadmium, Manganese, and Aluminum in the Blood of Pakistani Children with and without Autism Spectrum Disorder and Their Associated Factors* (2021)); Ex. 26, at 108:3-111:3 (Ritz MDL Tr.) (discussing same); *see also* Ex. 34, at 290:1-25, 292:17-294:2 (Aschner MDL Tr.).

Plaintiffs' experts also ignore studies showing that children who are introduced earlier to solid foods, including commercial baby foods, do *not* have an increased risk of subsequent autism diagnosis compared to children who receive their calories solely from breast milk and formula for a longer period of time. Rather, some studies suggest a *decreased* likelihood of autism diagnosis with earlier introduction of solid foods, including baby food. *See, e.g.*, Ex. 62 (Emond, et al., *Feeding Symptoms, Dietary Patterns, and Growth in Young Children With Autism Spectrum Disorders* (2010)) ("In infancy, the children subsequently diagnosed with ASD were more likely than controls to have late acceptance of solid food (P = .004)"); Ex. 66 (Campbell, et al., *Feeding Behaviors in Infants Later Diagnosed with Autism Spectrum Disorder* (2024)); Ex. 79 (Xiang, et al., *Association of feeding patterns in infancy with later autism symptoms and neurodevelopment: a national multicentre survey* (2023)) (finding that children with ASD have later introductions to foods and poorer acceptance of foods in infancy than typically developing children).

When shown examples of this literature at deposition, rather than admitting her oversight, Dr. Ritz immediately went into advocacy mode, speculating that the findings could reflect reverse causation—*i.e.*, that the infants who adopted solid foods first did so because they already had autism, even though undiagnosed. Ex. 26, at 134:3-136:2 (Ritz MDL Tr.). In other words, Dr. Ritz is willing to testify generally that children who eat Defendants' baby foods starting four to six months after birth can have autism *because they ate baby food.* But when shown data suggesting the opposite, her conflicting response is that the autism must have developed before the baby food eating began—it just had not yet manifested or been recognized. *Id*. at 132:14-134:21 (Ritz MDL Tr.) ("[W]hat I

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inadmissible. *See Daniels-Feasel v. Forest Pharms., Inc.*, No. 17 CV 4188-LTS-JLC, 2021 WL 4037820, at *5 (S.D.N.Y. Sept. 3, 2021).

suggested was that we don't know from this data alone because children's behavior and their food acceptance may already be a symptom for those 10 percent or 20 percent who did not accept solid foods. It may already be a symptom of their ASD."). Dr. Ritz's inconsistent testimony only highlights her repeatedly unscientific, result-oriented approach. *Kumho Tire Co.*, 562 U.S. at 152. "Result-driven analysis, or cherry-picking, undermine principles of the scientific method and is a quintessential example of applying methodologies (valid or otherwise) in an unreliable fashion." *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Practices and Prods. Liab. Litig. (No. II)*, 892 F.3d 624, 634 (4th Cir. 2018). Simply put, ignoring relevant evidence is "not good science." *In re Bextra and Celebrex Mktgs. Sales Practices and Prods. Liab. Litig.*, 524 F. Supp. 2d 1166, 1184 (N.D. Cal. 2007).

**5. Plaintiffs' Experts' Efforts to Support Their Opinions With Single Nutrient-Metals Interaction Studies Is Methodologically Flawed.**

Given the absence of any scientific studies that examine baby food and autism/ADHD risk, Plaintiffs' experts must find a way to bridge the analytical gap between the general heavy metal literature on which they rely and the ultimate opinions they offer about Defendants' baby foods. One of several ways they attempt to do so is by claiming that there is "insufficient evidence" that nutrients in food affirmatively *prevent* the "detrimental neurotoxic effects" of heavy metals like lead and arsenic. *See, e.g.*, Ex. 16, at 33 (Shapiro Rep.); Ex. 7, at 100, 103-104 (Gardener Rep.). Thus, the argument seems to go, if single nutrients like those that exist in food cannot, in isolation, prevent entirely lead or arsenic absorption from the environment, then Plaintiffs' experts can ignore their scientific duty to examine food as a complex mixture. This argument improperly shifts the general causation burden: It is Plaintiffs' burden to prove Defendants' baby foods *do* cause autism/ADHD, not Defendants' burden to show that food is protective. Moreover, the studies themselves are not methodologically reliable and cannot be used to bridge the gap between non-food studies and Defendants' baby food.

Plaintiffs' experts' attempts to link general heavy metal studies to Defendants' baby food are limited to biomarker studies of heavy metals and measures of single nutrient levels. What these papers investigate is whether single nutrients can reduce absorption or promote elimination of metals

in children at an elevated risk of environmental exposure. Some of these studies do so by comparing absorption of lead or arsenic in children who have adequate levels of a particular nutrient (like iron) to those who are deficient in that nutrient. Others consider whether supra-therapeutic doses of a nutrient can function as a chelating agent to help the body eliminate metals. *See* Ex. 1, at 26-27 (Ritz Rep.). For example, Dr. Ritz primarily relied upon Ruff, et al. (1996), a study which evaluated whether "moderately" lead poisoned children (blood lead levels between 25 and 55 mg/dL) experienced a greater reduction in blood lead level when supplemented with iron. *Id.* at 26 (also discussing Bouhouch, et al. (2016) and Farkhondeh, et al. (2022)). She also relied upon studies evaluating the impacts of calcium and Vitamin D in "high lead-burdened minority children." *Id.* at 27. Plaintiffs' epidemiology experts cite similar studies in their reports. *See, e.g.*, Ex. 4, at 34-35 (Hu Rep.) (citing to Ex. 95 (Wright, et al., *Association Between Iron Deficiency and Low-Level Lead Poisoning in an Urban Primary Care Clinic* (1999))) and Ex. 96 (Elias, et al., *Relationship Between Blood Lead Concentration and Nutritional Status Among Malay Primary School Children in Kuala Lumpur, Malaysia* (2007)); Ex. 7, at 94-104 (Gardener Rep.) (citing to Ex. 94 (Farkhondeh, et al., *Blood lead concentrations in children with iron deficiency anemia: a systemic review and meta-analysis* (2022))).

These single-nutrient biomarker studies cannot bridge the gap between general heavy metal studies and Defendants' baby food for at least three reasons. First, these studies do not consider food as a whole, but instead attempt to assess the impact of singular nutrients or vitamins such as iron, Vitamin D, or calcium, when food has dozens of nutrients and vitamins and other constituent parts. Food is a complex mixture, such that one cannot know how foods affect health just by looking at individual nutrients in isolation. As Dr. Hu admitted, one must look at food as a whole:

> Q. Why do you need to look at the combined effect of multiple nutrients at the same time?
>
> A. Well, I think from a public health point of view, I think that's what really counts most, which is, well, you know, what's—what's in food and what's the combination of things in food and what—how that might influence the primary relationships that you're interested in.

Ex. 29, at 187:14-22 (Hu MDL Tr.); *see also id*. at 189:7-190:6, 194:12-195:3; 195:9-14; Ex. 49, at 709:7-710:1 (Shapiro Landon Tr., Vol. II) ("I think that most of the studies are designed in such a

way that they're looking at the relationship of particular identified nutrients as opposed to all of the nutrients and all of the toxins to which someone could be exposed.").

The closest Plaintiffs' experts get to anything approaching a food opinion is discussion of a collection of literature that stands for the unremarkable proposition that consuming more rice (which is known to take up more arsenic than other foods in the growing process) leads to more arsenic excretion in urine. *See* Ex. 1, at 33 (Ritz Rep.) (discussing Ex. 97 (Karagas, et al., *Association of Rice and Rice-Product Consumption with Arsenic Exposure Early in Life* (2016))). But Dr. Ritz conceded at deposition that to assess arsenic body burden, "we need to take into account the *absorption* and the excretion and the *storage*." Ex. 26, at 177:6-25 (Ritz MDL Tr.) (emphasis added).

Second, the single nutrient studies that Plaintiffs' experts rely on evaluate reduction in blood and urine lead levels, but do not assess the impact of these nutrients on neurodevelopmental endpoints, much less autism and ADHD. *See, e.g.*, Ex. 43, at 52:19-53:8 (Ritz Landon Tr.) ("Q. Which nutrients, vitamins and minerals did you consider in determining whether food consumption has an impact on the neurological effects of heavy metals?. . . A. I -- I did not look at neurologic effects."), *id*. at 103:18-104:4 ("Q. Okay. Is it fair to say that you looked primarily at whether nutrients or vitamins could lower lead -- lead levels in blood when you concluded that food does not offset the harms of lead exposure? A. I think that's mostly what the studies had been doing, so that's what I would have been referring to… ."); *id.* at 104:1-4 ("Q: Did you look at any other mechanisms by which food can influence susceptibility to lead's effects other than lowering lead levels in the bloodstream? A. I don't think so."); Ex. 49, at 703:8-705:15 (Shapiro Landon Tr., Vol. II) ("I was looking for studies that were examining effects of various nutrients and their relationship to toxic exposures.").

Third, the designs of these studies, which investigate nutrient effects in children with high levels of environmental exposure to lead or arsenic, cannot speak to what happens at a dose and ratio of nutrients and metals found in food. Indeed, Plaintiffs' experts admit the studies show that higher levels of specific nutrients are positively correlated with *lower* levels of specific metals. *See* Ex. 29, at 169:14-170:17 (Hu MDL Tr.) (testifying that nutrient sufficiency can reduce the risk of

lead absorption); *id*. at 185:3-186:4 (testifying that nutrition can reduce lead burden on the body); Ex. 43, at 88:8-89:1 (Ritz Landon Tr.) (testifying that nutrients can impact absorption of lead); *id.* at 99:4-16 (testifying that nutrients can have some chelating effect on lead in the body); Ex. 48, at 353:1-10 (Shapiro Landon Tr., Vol. I) (conceding that heavy metal absorption "may be different based on the presence or absence of certain nutrients").

In short, these studies do not ask or answer the question of how trace amounts of lead or arsenic in Defendants' baby foods are capable of causing autism or ADHD. Plaintiffs cannot bridge the scientific gaps in their theory of causation with impermissible leaps that are built on data that do not address either the products or endpoints at issue. *See Joiner*, 522 U.S. at 146 (finding that expert opinions must be excluded when "there is simply too great an analytical gap between the data and the opinion proffered").

### B. Unable to Tie the Levels of Heavy Metals in Baby Foods to the Non-Food Literature on Which They Rely, Plaintiffs' Experts Resort to an Improper "Any Dose, Any Exposure, Any Window" Opinion.

Plaintiffs' experts' causation opinions must also be excluded because they are wholly untethered to basic scientific principles on the importance of dose. A central tenet of both toxicology and epidemiology is that "the dose makes the poison." Ref. Man. at 603; *see also* Ex. 36, at 49:14-16 (Shapiro MDL Tr., Vol. I) ("[T]he greater the degree of exposure, the greater the potential for toxicity"). That is, at some dose, any substance can be toxic—but conversely, not every dose of a given substance is toxic. To meet this basic scientific prerequisite, Plaintiffs proffer Dr. Jones to estimate the levels of arsenic and lead in a subset of Defendants' baby foods selected by Plaintiffs' lawyers, thereby providing the supposed evidentiary basis to allow the causation experts to conclude that those levels are sufficient to cause autism and ADHD. Setting aside the profound methodological flaws in Dr. Jones' analysis, which on their own warrant exclusion of her opinions (and opinions of all the subsidiary causation witnesses purportedly relying on her analysis), there is an equally fatal problem: Despite asserting in their reports their reliance on Dr. Jones' numbers, when it came time to defend this reliance, they quickly disclaimed doing so.

In their reports, the causation experts claim (in strikingly similar rote language) that the lead and arsenic levels in Dr. Jones's calculations are "consistent with" the levels associated with autism

and ADHD in the literature. Ex. 4, at 40 (Hu Rep.); Ex. 13, at 53 (Guilarte Rep.); Ex. 1, at 70 (Ritz Rep.). But even a cursory review shows that their only basis for this conclusion is their own say so. They did not attempt to tie the blood lead levels estimated by Dr. Jones to any literature showing those blood lead levels can cause autism or ADHD. On arsenic, they did even less. Unlike with lead, Dr. Jones did not even attempt to translate her estimates of the arsenic levels in Defendants' baby foods into an estimated level in children—not in urine, hair, or blood—and thus, did not even try to connect those levels of arsenic in Defendants' foods to any literature.

Thus, when pressed in deposition to elaborate on how they were relying on Dr. Jones' estimates, Plaintiffs' experts demurred and said that those estimates were not necessary to their ultimate causation opinions because there is "no safe level" of lead or arsenic. *See, e.g.*, Ex. 36, at 14:13-21; 63:15-23 (Shapiro MDL Tr., Vol. I). In other words, the experts seem to be retreating to claim that *any* dose of lead or arsenic is capable of causing autism or ADHD, irrespective of the levels estimated by Dr. Jones. Of course, that is not what FDA and the CDC meant when they stated there is "no safe level" of heavy metals. They simply meant that the level below which there are no health effects of any kind over any length of exposure is unknown. *See* FDA, Closer to Zero: Reducing Childhood Exposure to Contaminants from Food (2025), Closer to Zero: Reducing Childhood Exposure to Contaminants from Foods | FDA. Yet, that is precisely Plaintiffs' experts' position. Ex. 36, at 151:20-152:4 (Shapiro MDL Tr., Vol. I) (admitting that "you could imagine some circumstance in which" a child's exposure to one carrot with one microgram of arsenic caused autism). And indeed, Plaintiffs' experts' willingness to accept *any* dose as being "consistent with" the literature is evident in the fact that each of them continued to find Dr. Jones's dose calculations sufficient, notwithstanding that she had three different calculations, with each one being lower than the prior calculation. *Compare* Ex. 21 (Jones Rep.), *with* Ex. 23 (Jones Rebuttal Rep.), *and* Ex. 24 (Jones Amended Rebuttal Rep).

**1. Plaintiffs' Experts Ignore Dose Altogether or Fail to Engage In Any Methodology to Attempt to Link Their Exposure Expert's Calculations to the Literature.**

Plaintiffs understand that at this stage of the litigation they are required to demonstrate that lead and arsenic *at in the levels contained in each Defendants' baby foods* and in the baby foods

themselves can cause autism and/or ADHD. That's why they retained Dr. Jones. Her charge was to calculate the levels of lead and arsenic for each Defendant in the hypothetical menus provided to her, translate the levels of lead she calculated in the products into a blood lead level, and provide those estimates to the causation experts so they could answer the question at the heart of this case: can the levels of lead and arsenic in each Defendants' baby foods cause autism and ADHD?

That is not what happened. The causation experts did not engage in any analysis to link the exposure levels calculated by Dr. Jones to the epidemiologic studies they claim support their opinions. Drs. Gardener, Shapiro, and Aschner failed specifically to address the issue of dose in their expert reports. Most strikingly, when asked about how dose and blood lead levels informed his causation opinion, Dr. Hu disclaimed doing any such analysis:

> Q. What range of changes in blood lead level do you consider to be meaningful or significant changes in terms of autism risk?
>
> MS. FORGIE: Objection. Incomplete hypothetical.
>
> THE WITNESS: **I don't have an opinion on that.**
>
> Q. What range of changes in blood lead level do you consider to be meaningful or significant changes in terms of ADHD risk?
>
> MS. FORGIE: Objection. Incomplete hypothetical.
>
> THE WITNESS: **I don't have an opinion on that either.**

Ex. 29, at 231:4-18 (Hu MDL Tr.) (emphasis added). Dr. Ritz similarly took a pass. Despite saying in her report that she did so, Dr. Ritz testified that she "never made [the] comparison" between the range or ranges Dr. Jones found and the ranges seen in various study populations of exposure. Ex. 26, at 180:21-181:9 (Ritz MDL Tr.). For his part, Dr. Guilarte admitted he had no opinion on a minimum dose and duration of exposure to lead or arsenic that is necessary to cause autism or ADHD. *See* Ex. 35, at 85:1-8, 333:7-20 (Guilarte MDL Tr.); *see also* Ex. 13, at 53 (Guilarte Rep.).

Instead, Plaintiffs' experts "just extrapolated." Ex. 26, at 180:21-181:9 (Ritz MDL Tr.). Dr. Ritz inferred that Dr. Jones' "dose" calculations were sufficient to cause autism/ADHD based on the FDA and CDC statements that "no safe level" of lead exists. *Id.* ("A. You can extrapolate. And that's based on what FDA also says and CDC says, that there's no safe level, so we're allowed to extrapolate.") Dr. Hu extrapolated, without basis, from studies relating to cognition (small

changes in IQ), not an autism diagnosis. Ex. 29, at 82:13-86:10 (Hu MDL Tr.) ("There is no threshold that I'm aware of that's been identified below which, you know, lead could not be expected to have a deleterious effect. That's certainly true for cognition. I don't think anybody's tried to look at that with respect to autism.").

Plaintiffs are thus unable to show that *any* of the estimated levels of arsenic and lead that may be present in Defendants' baby foods (that Dr. Jones tried three times to calculate) are capable of causing autism or ADHD.

**2. Plaintiffs' Experts Retreat to an Unreliable "No Safe Dose" Opinion—That Eating One Jar of Carrots Can Cause Autism.**

Faced with an inability to show that Dr. Jones' outputs of lead and arsenic levels can cause autism or ADHD, Plaintiffs' experts fall back on the opinion that there is "no safe level" of lead or arsenic and therefore any level can cause autism and ADHD. In other words, according to the experts' reasoning, eating only a single pouch of baby food containing trace levels of lead, or one carrot from the grocery store with detectable arsenic, could cause autism/ADHD. As Dr. Shapiro testified: "I also don't have any studies that show that carrots don't cause ASD and ASD—ADHD. So it certainly—you know, if you're talking about, like, the study of the particular food and its relationship to a particular outcome, I could equally claim that as far as I know, carrot consumption causes autism." Ex. 36, at 125:10-126:13 (Shapiro MDL Tr., Vol. I); *id.* at 151:20-152:4 (admitting that "you could imagine some circumstance in which" a child's exposure to one carrot with one microgram of arsenic caused autism).

As the experts well know, however, the concept of "no safe level" of heavy metal exposure is a precautionary regulatory principle—not a conclusion that exposure to *any* level of lead or arsenic can cause harm, including autism or ADHD. Nor could it be the logical meaning, given that every person inhales and ingests heavy metals throughout their lifetime. As Plaintiffs' experts agree, at least some traces of these metals can be found virtually everywhere. *See* Ex. 37, at 338:16-339:8 (Shapiro MDL Tr., Vol. II) ("[T[here is always some nonzero amount of—as far as I know, always some nonzero amount of lead and arsenic in foods. And so I think that saying that one should not eat food with any lead or arsenic would be equivalent to saying you shouldn't eat food."); Ex. 29,

at 278:19-279:6 (Hu MDL Tr.) (stating that lead is "present not only in the earth's crust, but because of the use of lead in industry and consumer products, there's a little bit of lead just about everywhere."). Carrying the experts' opinions to their logical conclusion, they would say that just living in the world causes autism.

For good reason, courts routinely reject "no safe level" opinions as irrelevant to the question of causation. *See, e.g.*, *In re Zantac*, 644 F. Supp. at 1241; *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1241 (11th Cir. 2005); *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278 (5th Cir. 1998) (excluding expert testimony which "offered no scientific support for his general theory that exposure to toluene solution at any level would cause RADS"); *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1166 (E.D. Wash. 2009) (excluding testimony that "any amount of exposure" to chemical "can cause" cancer); *Krik v. Exxon Mobil Corp.*, 870 F.3d 669, 677 (7th Cir. 2017) (noting that "more than thirty other federal courts and state courts have held that this cumulative/'any exposure' theory is not reliable"); *Sutera v. Perrier Grp. Of Am. Inc.*, 986 F. Supp. 655, 666 (D. Mass. 1997) ("[T]here is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique[.]").

### C. There Is Too Great of an Analytical Gap Between the Non-Food Data Plaintiffs' Experts Rely Upon and the Opinions They Offer.

Because no scientific literature supports (or even examines) an association between consumption of baby food or other healthy foods in infancy/toddlerhood with autism or ADHD, Plaintiffs' experts attempt to substantiate their "any dose, any route" causation opinions by pointing to a body of literature evaluating lead and arsenic exposure *generally* and neurodevelopmental effects *broadly defined*. But this literature bears little resemblance to the general causation question before this Court: It addresses different sources of exposure (unknown or other sources vs. foods), different windows of exposure (prenatal periods or post-baby-food use periods vs. the period during which children eat baby food), different populations (ex-U.S. vs. U.S.), and endpoints that are different from those used to diagnose autism and ADHD in real-world clinical practice (non-specific symptoms that may or may not be associated with those diagnoses vs. diagnosed autism/ADHD).

Courts must exclude expert opinions where there is "simply too great an analytical gap" between the studies relied upon "and the opinion proffered." *Joiner*, 522 U.S. at 146. When an expert relies on studies conducted under conditions materially different from those at issue in the litigation, courts must assess whether the literature cited is sufficiently analogous to the facts of the case such that the inference drawn by the expert is reliable. *Id.* (excluding testimony based on studies that lacked relevance to the plaintiff's condition). Improper extrapolation, "[t]hat is, leaping from an accepted scientific premise to an unsupported conclusion," is a classic Rule 702 "red flag." *Downs v. Perstorp Components, Inc*., 126 F. Supp. 2d 1090, 1125 (E.D. Tenn. 1999). Importantly, the party proffering the evidence must demonstrate that the method of extrapolation is generally accepted. *See Schudel v. GE*, 120 F.3d 991, 997 (9th Cir. 1997) (concluding that an expert improperly extrapolated from studies regarding the neurotoxic effects from acute exposure to chronic exposure because there was no showing that such extrapolation was scientifically acceptable). In addition, to be admissible, an expert's opinion must be based on scientifically valid principles and must "fit" the facts of the case—that is, it must "speak[] clearly and directly to an issue in dispute in the case, and . . . will not mislead the jury." *Daubert v. Merrell Dow Pharms*, 43 F.3d 1311, 1321 n.17 (9th Cir. 1995). To "fit" the facts of the case, scientific literature must be generalizable to the facts at issue. *See Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1397 (D. Or. 1996) must still exclude the evidence if it does not 'fit' the matters at issue in the case.").

Here, Plaintiffs' experts engage in *no methodology* to establish that the literature they cite can be reliably extrapolated to the question of whether baby food causes autism or ADHD—or even to the question of whether exposure to *heavy metals generally* during early postnatal life can cause diagnosed autism or ADHD. Thus, while Plaintiffs' experts make much of the fact that there are dozens, or even hundreds, of heavy metal studies cited in their reports, Rule 702 is not a mere counting exercise: the experts fail to acknowledge the gaping "analytical gaps" between those studies and the ultimate question in this case and the opinions they offer to answer. For numerous reasons, this body of literature cannot reliably answer the general causation question before the Court.

Sections III(A) and III(B), *supra*, describe the analytical leaps Plaintiffs' experts take in

rendering conclusions about baby food from studies that do not involve food or the doses to which children are exposed to heavy metals through baby food. As described in the sections immediately below, the literature cited by Plaintiffs' experts is disconnected from the issues in this litigation because it does not consider the correct endpoints (i.e., autism and ADHD), the developmental window of exposure, or the population exposed.

**1. Plaintiffs' Experts Rely on Literature that Does Not Study Diagnosed Autism/ADHD Endpoints.**

The first problem is that Plaintiffs' experts rely on literature that does not consider the actual injuries alleged by every plaintiff in this case—diagnosed autism and ADHD. And the experts applied no methodology that would enable them to reliably bridge the gap between the non-diagnostic outcomes in studies they cite and their ultimate opinions that trace amounts of heavy metals in Defendants' baby food can cause autism and ADHD.

Under Rule 702, every causal inquiry requires determining "whether exposure to a substance for which a defendant is responsible . . . at the level of exposure alleged by plaintiffs, is capable of *causing a particular injury or condition*." *In re Hanford Nuclear Rsrv. Litig. v. E.I. Dupont*, 292 F.3d 1124, 1133 (9th Cir. 2002) (emphasis added). In the Tylenol MDL, Judge Cote considered claims that Tylenol is associated with autism and ADHD and recently underscored the need for studies with actual diagnoses of these conditions as an endpoint to support a causation opinion. *See In re Acetaminophen - ASD-ADHD Prods. Liab. Litig.*, 707 F. Supp. 3d 309, 364 (S.D.N.Y. 2023). There, the court excluded the plaintiffs' experts in part for failing to "confine themselves to studies that relate to diagnoses of ASD and ADHD" and relying on "studies of symptoms that reflect many endpoints relevant to [neurodevelopmental disorders] generally, including to ASD and ADHD." *Id.* The court noted that such an "unstructured approach . . . permitted cherry-picking, allowed a results-driven analysis, and obscured the complexities, inconsistencies, and weaknesses in the underlying data." *Id.*

Conclusions about autism and ADHD can be drawn only from literature regarding diagnosed autism and ADHD, because diagnosed autism and ADHD are distinct neurodevelopmental conditions with specific requirements set forth in the DSM-V. Ex. 57, at 99:18-23 (Jan. 31, 2022,

Shapiro N.C. Sargon Tr.) ("Q. There is a difference between autism and generalized neuro-developmental symptoms; correct? A. Well, autism is a specific constellation of symptoms, so not all children with neurodevelopmental issues will have a diagnosis of autism.") Having "symptoms" that may be part of autism/ADHD or co-occur in autism/ADHD is not the same as having a formal diagnosis. Ex. 35, at 68:17-69:10 (Guilarte MDL Tr.); Ex. 37, at 296:10-296:16 (Shapiro MDL Tr., Vol. II) ("Q. Okay. A child whose symptoms do not cause clinically significant impairment would also not meet the criteria for a diagnosis of autism. Correct? A. That is a—that is a requirement for the clinical diagnosis, yes."); Ex. 57, at 103:1-5 (Jan. 31, 2022, Shapiro N.C. Sargon Tr.) (agreeing that unless a child meets "all" of the criteria for DSM-5 "they don't by definition have autism."). Symptoms of autism and ADHD are non-specific and may be associated with diagnoses other than autism and ADHD. *See, e.g.*, Ex. 37, 295:23-296:9 (Shapiro MDL Tr., Vol. II) ("Q. Okay. A child who only exhibits behavioral problems would not meet the criteria for a diagnosis of autism under the DSM-5-TR criteria. Correct? A. They would not meet those criteria, yes. Q. And those children also could have some form of neurodevelopmental disorder or dysfunction. Correct? A. They could, yes.")

For example, some children with autism can have low IQ, but low IQ can also be associated with a host of other conditions, and many children with autism have perfectly normal (or even high) IQ. Consistent with this, Dr. Hu acknowledged that IQ is *not* an appropriate endpoint for studying autism/ADHD diagnosis. *See* Ex. 29, at 154:13-18 (Hu MDL Tr.) ("So I—you know, autism really is more of a behavioral diagnosis than an intelligence diagnosis. So I would not feel confident in providing an overall opinion about whether autistic kids are, in general, intelligence impaired or not."); *see also id.* at 154:20-24 ("IQ is not part of the criteria for diagnosis ASD."). Dr. Guilarte admitted that "*to reach reliable opinions about causation of ASD, you need to look at diagnosed ASD*" and "*the same is true for ADHD*." Ex. 35, at 71:18-72:4 (Guilarte MDL Tr.) (emphasis added).

Despite at least some Plaintiffs' experts admitting that conclusions about autism and ADHD can be drawn reliably only from studies involving diagnosed autism and ADHD, all the experts rely on studies not involving those outcomes. Instead, they rely on studies that examine whether an association exists between heavy metal exposure and a *variety* of outcomes in addition to diagnosed

autism/ADHD—including IQ, behaviors, symptoms, teacher or parent/caregiver surveys, and autism/ADHD screening tools. *See, e.g.*, Ex. 1, at 19 (Ritz Rep.) ("I did not limit my review to studies that considered only diagnoses of ASD and/or ADHD. Instead, I also considered studies in which the endpoint(s) was/were behavior(s) and/or symptom(s) associated with ASD and/or ADHD, but where no formal diagnosis had been made."); *see also* Ex. 29, at 273:4-24 (Hu MDL Tr.); Ex. 46, at 200:1-24 (Gardener Landon Tr.); Ex. 34, at 114:8-18 (Aschner MDL Tr.); Ex. 35, at 23:12-25 (Guilarte MDL Tr.); Ex. 36, at 17:7-18:10 (Shapiro MDL Tr., Vol. I).[10]

For example, all of Plaintiffs' epidemiologists rely on Kim et al. (2016), which excluded from its endpoint any children with an actual autism diagnosis and included children based on responses to two questionnaires, the Autism Spectrum Screening Questionnaire ("ASSQ") and the Social Responsiveness Scale ("SRS"). *See* Ex. 85 (Kim, et al., *Low-Level Lead Exposure and Autistic Behaviors in School-Age Children* (2016)). Based on the ASSQ, for example, a child might be included in the "autism" group because the child is regarded as an "eccentric professor," has restricted intellectual interests, accumulates facts on certain subjects, has a different voice or speech, is surprisingly good at some things and not good at others, makes naïve and embarrassing remarks, is bad at games, is clumsy, is bullied, or does not have a best friend.[11] *See Autism Spectrum Screening Questionnaire (ASSQ)*, Autism Spectrum Screening Questionnaire (ASSQ) - Psychology Tools (last visited Sept. 26, 2025). Several experts also relied on Alampi et al. (2021), which included children in the "autism" group based on responses to the SRS, such as whether the child

[10] In prior baby food cases, Plaintiffs' experts Drs. Ritz, Shapiro, and Gardener stated that the general causation question was whether heavy metals, including in baby food, can cause autism/ADHD. In this litigation, they offer a far more convoluted opinion, presumably because the literature on which they rely does not address the outcome at hand—autism/ADHD diagnosis. *See, e.g.*, Ex. 7, at 6 (Gardener MDL Rep.) (framing the general causation question as whether doses of heavy metal from consumption of a hypothetical menu of commercial baby food "can interfere with early neurodevelopment and result in a set of behaviors that can be diagnosed as" autism/ADHD).

[11] ASSQ, *available at* https://novopsych.com/wp-content/uploads/2025/01/Autism-Spectrum-Screening-Questionnaire-ASSQ-blank-form-pdf-survey-questions-items.pdf; *see also* Ehlers, S., Gillberg, C., & Wing, L. (1999). A screening questionnaire for Asperger syndrome and other high-functioning autism spectrum disorders in school age children. Journal of Autism and Developmental Disorders, 29(2), 129-141, *available at* https://doi.org/10.1023/a:1023040610384.

avoids eye contact, avoids physical contact, and has difficulty sharing or trouble expressing empathy.[12] *See* Ex. 90 (Alampi, et al., *Association Between Gestational Exposure to Toxicants and Autistic Behaviors Using Bayesian Quantile Regression* (2021)); *Social Responsiveness Scale-2 (SRS-2) Profile Sheet*, Attachment 8 g Social Responsiveness Scale (Child) (last visited Sept. 26, 2025). All these qualities may be associated with autism or may be associated with a host of other conditions—or no condition at all. *See* Ex. 57, at 103:1-5 (Jan. 31, 2022, Shapiro N.C. Sargon Tr.) ("Q. But just so I understand your testimony, did I hear you to say that just because you're positive on one of those screening mechanisms, that doesn't equate with a diagnosis of autism; correct? A. That's true. Autism is a clinical diagnosis so you need to put together multiple sources of information."); Ex. 51, at 144:16-145:8 (Shapiro N.C. Tr., Vol. I) (agreeing that reductions in intelligence, behavioral problems, symptoms of attention deficit, hyperactivity, and cognitive control are symptoms not specific to autism).

Plaintiffs' experts have no way of reconciling their reliance on these studies with their acknowledgment that diagnosed autism and ADHD are, in fact, the proper endpoints used to evaluate whether an exposure causes these conditions. And they did not conduct any analysis or apply any disclosed methodology to attempt to support an extrapolation to endpoints that are not validated surrogates for these conditions. For example, Plaintiffs' experts did not conduct *any* analysis to determine whether the purported impact of heavy metal exposure varied depending on the particular outcome studied—e.g., diagnosed autism vs. "autistic behaviors"—in terms of the strength of any supposed association. Nor did they conduct any analysis to determine whether the studies they rely on use definitions of autism and ADHD that "have been demonstrated to be highly predictive" of an actual autism or ADHD diagnosis. *See* Ex. 29, at 25:24-28:6 (Hu MDL Tr.); *see, e.g.*, Ex. 26, at 34:20-35:1 (Ritz MDL Tr.) (testifying that she did not compare results of studies that examined autism symptoms versus an autism diagnosis). Finally, for studies that evaluated only behaviors and symptoms, the experts did no independent analysis of whether the particular behavior(s) or symptom(s) studied are correlated with autism or ADHD—as opposed to some other

[12] SRS-2, *available at* https://autismspectrumtest.com/social-responsiveness-scale/.

neurological or cognitive endpoint not at issue in this litigation. *See*, *e.g.*, *id.* at 34:20-35:1 (failing to compare the results of studies that only examined symptoms versus studies that looked at the diagnosis of autism); *id.* at 34:2-19 (failing to evaluate whether estimated measures of association differ depending on whether studies looked at symptoms versus autism diagnosis); Ex. 43, at 52:19-53:8 (Ritz Landon Tr.) (admitting she "did not look at neurologic effects").

This Court should exclude the experts' general causation opinions for failing to "confine themselves to studies that relate to diagnoses of ASD and ADHD" and relying on other endpoints, like IQ, that the experts recognize are not necessarily highly correlated with autism or ADHD. Plaintiffs' experts provide no methodology to bridge the gap between the studies evaluating non-diagnostic outcomes (behaviors, symptoms, IQ, and other non-specific outcomes) and their ultimate conclusion that baby food containing trace levels of lead or arsenic can cause autism/ADHD. This failure renders their opinions inadmissible under Rule 702. *See In re Acetaminophen - ASD-ADHD Prods. Liab. Litig.*, 707 F. Supp. 3d at 334 (rejecting this same "transdiagnostic approach" that "sweeps into their analyses (and conclusions) ASD, ADHD and other neurodevelopmental disorders"); *see also Joiner*, 522 U.S. at 146 (finding that a district court need not "admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered").

**2. Plaintiffs' Experts Unreliably Seek to Extrapolate from All Time Periods of Exposure to the Neurodevelopmental Window When Children Eat Baby Food.**

The second problem is that Plaintiffs' experts rely heavily on studies involving heavy metal exposures during developmental windows when baby food is not consumed, including studies related to *prenatal* maternal exposures and studies examining heavy metal exposures in children who are well past the age of consuming baby food.

As a threshold matter—and most importantly—the prenatal and too-late-postnatal studies Plaintiffs' experts rely on cannot support their causation opinions because those studies largely fail to show *any* positive association (reliable or otherwise) between heavy metal exposure and autism or ADHD. *See infra* Section III(D). Beyond that, however, Plaintiffs' experts cannot reliably presume, as they do here, that an exposure during one developmental window (i.e., prenatal) will

create the same effect in another developmental window (i.e., time period when a child eats baby food)—particularly considering that the physiology of neurodevelopment differs greatly during these different time periods. *See, e.g.*, Ex. 45, 166:4-167:1 (Ritz Landon Tr.) ("Q. How is cell division different pre- and post-birth? A. Well, it still happens post-birth, but post-birth, it's mostly maturation and synaptic development, pruning and—and also, yeah, synapse forming. Q. And what happens pre-birth? A. Pre-birth, everything happens, basically. The brain, you know, develops cells, microglia. It doesn't yet form the myelin sheaths. So those are also quite important, and they are mostly end of pregnancy, early childhood.")

Exposure to heavy metals through commercial baby food occurs at a unique time: during infancy and possibly into the first two to three years of life. As even Plaintiffs' experts admit, timing of exposure is critical when evaluating causation related to heavy metals because lead and arsenic exposures generally can have different effects on a child's body depending on when the exposure takes place. *See* Ex. 26, at 48:3-10 (Ritz MDL Tr.) ("Q. Okay. And if I'm understanding you correctly, is it fair to say that lead exposure can have different effects depending on the timing of that exposure? A. Correct. Q. Okay. And is the same true for inorganic arsenic? A. Yes."); *see also id.* at 174:7-11 ("I mean, we talked about timing and, you know, being more sensitive when you're younger than maybe when your brain is more mature."); Ex. 45, at 177:20-178:3 (Ritz Landon Tr.) ("Q. Can you identify for me studies examining gene-environment interactions that found significant associations with autism for postnatal environmental exposures that occurred at age six months of age or older? . . . A. Yeah, I don't think those studies have been done, but they should be done."). Indeed, in her work *outside* the courtroom, Dr. Ritz has conducted research that distinguishes the prenatal and postnatal effects of other environmental exposures. *Id.* at 269:15-270:4 ("Q. Before reaching your opinions in the case, did you conduct any analysis that stratified by age range when exposure was measured? . . . A. In children? Q. Yes. A: I think we did that in my air pollution studies, actually. Q. Have you seen that done in any of the studies evaluating the potential relationship between lead or inorganic arsenic and autism? A. I don't think they had that luxury of multiple measurements."); *see also id.* at 164:6-12 (explaining that in her work on air pollution, she has stratified based on age).

Timing is particularly critical when discussing neurodevelopmental outcomes. As Dr. Ritz conceded, "there are different parts of the brain that mature at different times," and neurodevelopment can vary from *month to month*. Ex. 26, at 45:15-46:8, 219:24-215:10 (Ritz MDL Tr.); *see also* Ex. 36, at 229:11-230:22 (Shapiro MDL Tr., Vol. I) (stating that key neurodevelopmental stages vary significantly by age group). Dr. Ritz also noted that various parts of the brain—"the motoric system, the language system, the social communication, executive function . . . develop at different times." Accordingly, "depending on what the timing of the exposure is, there are different effects on this very, very complex system and its development." Ex. 26, at 47:6-48:2 (Ritz MDL Tr.).

As Dr. Ritz admitted, the relevant period for the causation question in this litigation is "during neurodevelopment," meaning "after the baby is born and before the diagnosis is made." Ex. 26, at 43:19-25 (Ritz MDL Tr.); *see also* Ex. 35, at 83:25-84:4 (Guilarte MDL Tr.) ("Q. And in order to determine if a substance can cause ASD postnatally, one should look at studies of postnatal exposure, correct? A. Correct. Q. Okay. And the same is true for ADHD obviously, correct? A. Correct.") (emphasis added). And as Dr. Shapiro testified, the most obvious risk factors that contribute to a child's development of autism occur *prenatally*. Ex. 37, at 266:4-267:1 (Shapiro MDL Tr., Vol. II). This makes it critical to differentiate between prenatal and postnatal exposures when assessing potential causes of autism and ADHD.

Plaintiffs' experts nevertheless rely on studies involving *prenatal* exposures to heavy metals. For example, Plaintiffs' experts cited studies that evaluated (1) maternal blood samples collected *during pregnancy* to assess exposure to various heavy metals, and (2) amniotic fluid samples collected *during pregnancy* to assess exposure to lead and arsenic. *See* Ex. 16, at 25, 27, 29, 34 (Shapiro Rep.) (relying on Skogheim et al. 2021); Ex. 7, at 49, 60 (Gardener Rep.) (relying on Long et al. 2019); *see also* Ex. 33, 97:16-100:17; 221:1-225:4 (Gardener MDL Tr.). And when they consider scientific literature addressing *postnatal* exposure, Plaintiffs' experts fail to limit their analysis to the effect of exposure to heavy metals during the period when baby food is typically consumed—instead relying on studies involving children who are many years past the age at which

autism is generally understood to have manifested. *See, e.g.*, Ex. 43, at 285:2-8 (Ritz Landon Tr.) (conceding that studies do not stratify metal exposure by age or adjust for time period).

Even if those studies showed a reliable association between heavy metal exposure and autism/ADHD (which they do not, *see infra* Section III.D), Plaintiffs' experts offer no reliable methodology that would allow them to apply the findings of studies assessing the effects of exposure outside of the window in which children eat baby food to the effects that they conclude occur when exposure is within that window. *See, e.g.*, Ex. 43, at 279:9-285:8 (Ritz Landon Tr.) (failing to explain discrepancy in systematic review that purportedly showed a correlation between prenatal exposure to heavy metals and increased risk of autism, but not during post-natal exposure). This is critical, as "[c]onfidence in the appropriateness of an extrapolation cannot come from the experiment itself. It comes from knowledge about outside factors that would or would not affect the outcome." Ref. Manual at 223. Yet various Plaintiffs' experts did not do *any* work to understand the "outside factors"—in this case, the time window during which autism is understood to develop and the differing effects of heavy-metal exposure during various developmental windows, which could influence the outcome. Dr. Gardener, for example, testified that the distinction between prenatal versus postnatal exposure as it relates to autism and ADHD causation does not need to be evaluated. *See* Ex. 33, at 228:4-18 (Gardener MDL Tr.); *see also* Ex. 29, at 220:15-221:4 (Hu MDL Tr.) (explaining "[t]here hasn't been a lot of research that's been done that could rigorously compare" the prenatal and postnatal windows of exposure). As a result, Dr. Gardener did not do ***any*** "comparison of—of exposures prenatally versus postnatally." Ex. 46, at 198:13-20 (Gardener Landon Tr.); *accord* Ex. 34, at 184:12-17 (Aschner MDL Tr.) (showing that Dr. Aschner "did not attempt to stratify studies by whether they controlled for just postnatal exposure or not"). And while Dr. Hu stated that "in theory" lead can have different effects on a child's body prenatally compared to postnatally, he did not know if prenatal or postnatal lead exposure bears a greater risk of ADHD because he did not "review[] the literature from that . . . particular lens." Ex. 29, at 224:13-19, 254:7-16 (Hu MDL Tr.).

Dr. Hu nonetheless attempted to justify relying on prenatal studies by stating that "[f]ew of the epidemiological studies of the relationship between lead exposure and the development of

ADHD or ASD… have exposure information (e.g., blood lead levels) taken specifically during the 1st year of life (vs. prenatally, or at other time points)." Ex. 4, at 35 (Hu MDL Rep.); *accord* Ex. 29, at 224:13-19 (Hu MDL Tr.) (noting that he did not have "a lot of evidence in which to compare the distinctions or any differences between those effects"). But that does not excuse improper reliance on data from the wrong neurodevelopmental window when a human's physiology is much different, or Plaintiffs' experts' attempted extrapolation from studies involving prenatal exposures. It simply confirms that there is no reliable scientific evidence addressing the actual general causation question here. *See Perry*, 564 F. Supp. 2d at 467-68 ("[T]he non-existence of good data does not allow expert witnesses to speculate or base their conclusions on inadequate supporting science.").

Similarly, Plaintiffs' experts did not evaluate whether the effects of postnatal heavy metal exposure on autism/ADHD vary by age *at all*, or even attempt to define the critical window for heavy metal exposure as it relates to the development of autism/ADHD. Dr. Ritz could not determine whether the supposed risk of autism/ADHD from heavy metal exposure varied at all in children ages zero to age ten. *See* Ex. 43, at 43:1-11 (Ritz Landon Tr.); *see also id.* at 42:7-24 (refusing to differentiate increased risk of autism between pre- and postnatal exposure to heavy metals); *id.* at 268:20-269:3 (conceding that she cannot weigh the impact of pre- and postnatal heavy metal exposure because she did not do or find "a study that actually evaluates the exposures throughout and estimates effects"). Nor could Dr. Gardener, who did not even consider the relevant time window for autism development when evaluating whether exposure at ages zero to three years old can cause autism. Ex. 46, at 207:9-15, 208:6-22 (Gardener Landon Tr.).

An expert cannot simply conclude, as Plaintiffs' experts do here, that exposure to heavy metals during any time window when a child eats baby food can cause autism/ADHD without a reliable methodology supported by reliable data from that time window. *See Olive v. Gen. Nutrition Ctrs., Inc.*, 30 Cal. App. 5th 804, 819 (2018) ("An expert may not base his or her opinion upon a comparison of matters that are not reasonably comparable."). Rule 702 forbids Plaintiffs' experts' unsupported guesswork and speculation that, because heavy metal exposure at some point can cause autism or ADHD, heavy metal exposure during the neurodevelopmental period when children typically eat baby food has the same effects on the anatomical structures that matter. *See Rosen*, 78

F.3d at 319 ("[T]he court room is not the place for scientific guesswork, even of the inspired sort."). Because Plaintiffs' general causation opinions cannot bridge this analytical gap, their opinions on whether exposure to heavy metals during the developmental window when baby food is consumed can cause autism/ADHD are inadmissible.

**3. Plaintiffs' Experts Unreliably Extrapolate from Studies of Non-U.S. Populations with Massive Heavy Metal Exposures.**

The third problem is that Plaintiffs' experts place heavy weight on studies that fail to examine the relevant population at issue in this litigation: U.S. children. For example, Dr. Guilarte acknowledged that "most of the heavy metal epidemiological studies in [his] report are from non-western and/or non-industrialized nations such as Egypt, India, Africa, Saudi Arabia, China," that "children's exposures to heavy metals in these countries are much higher than children's exposures in the U.S.," and that "the prevalence of high lead levels in those countries is much higher." Ex. 35, at 121:6-122:1 (Guilarte MDL Tr.).[13] Dr. Guilarte further admitted that "the heavy metal studies that are most relevant to the US population . . . are studies from the United States." *Id.* at 128:23-129:2; *see also id.* at 131:25-132:5 (agreeing that "geographic location globally may influence observed associations between heavy metals and ASD").

Here, however, Plaintiffs' experts rely on the reported results from numerous studies and meta-analyses that evaluate heavy metal exposures and autism/ADHD (or other neurodevelopmental behaviors and symptoms) in populations outside the United States. To take one example, Plaintiffs' causation experts rely on Stojsavljevic et al. (2023), a meta-analysis that included studies from populations across the world that examined whether there is a relationship between autism and blood lead levels. *See* Ex. 88 (Stojsavljevic, et al., *Does Lead Have a Connection to Autism? A Systematic Review and Meta-Analysis* (2023)). For many of the ex-U.S. studies included in that meta-analysis, the study participants' blood lead levels differ dramatically from the blood lead levels seen in virtually all U.S. children. One study from Iraq included in the

[13] Indeed, Dr. Guilarte has published on the "exponentially higher lead levels" in children in developing countries, as compared to children in the U.S. Ex. 35, at 125:11-17 (Guilarte MDL Tr.).

meta-analysis, Alawad et al. (2019), showed that the *controls* evaluated in that study—meaning children *without* autism—had mean blood lead levels of almost *17.4* ug/dL. *See* Ex. 89 (Alawad, et al., *Lead Among Children With Autism In Iraq* (2019)). And the *average* blood lead level for *controls* across all the studies included in the Stojsavljevic meta-analysis was 3.8 ug/dL. Data published by the EPA, however, shows that median blood lead levels of U.S. children ages 1 to 5 are approximately *0.6 ug/dL*[14]—meaning that U.S. children have average blood lead levels approximately six times lower than the average control child included in this meta-analysis. Plaintiffs' experts did not even attempt to explain how the results from this meta-analysis can be used to reach *any* reliable conclusions about the effects of lead exposures in U.S. children.

As another example, Ding et al. (2023)—one of the most recently published meta-analyses to date evaluating the association between heavy metals and autism, on which Drs. Hu, Ritz, and Gardener all rely—included a total of 41 studies that measured lead exposure in a number of different countries. *See* Ex. 64 (Ding, et al., *Association between heavy metals exposure (cadmium, lead, arsenic, mercury) and child autistic disorder: a systematic review and meta-analysis* (2023)). The Ding meta-analysis explicitly evaluated the results of studies in North America, as opposed to different populations, and concluded that lead levels were *not* statistically significantly higher in children with autism *in North America* as compared to controls. *See, e.g.*, Ex. 29, at 105:16-108:8 (Hu MDL Tr.) (testifying that when stratified by region, lead levels were not statistically significantly higher in children with autism in North America); Ex. 43, at 306:17-22 (Ritz Landon Tr.) (discussing Ding et al. (2023) and agreeing that "in the studies in North America, the authors did not find a statistically significant relationship between exposure to either lead or arsenic and the risk of autism"). Yet, Plaintiffs' experts rely on Ding's findings in non-U.S. populations, which did purport to show a statistically significant increase in lead levels in children with autism as compared to children without autism, as well as the foreign studies included in that meta-analysis, as a key

[14] EPA, Biomonitoring – Lead (2023), Biomonitoring - Lead | US EPA ("The median concentration of lead in the blood of children between the ages of 1 and 5 years dropped from 15 µg/dL in 1976–1980 to 0.6 µg/dL in 2017–March 2020, a decrease of 96%.").

piece of evidence supporting their general causation opinions.[15]

To be clear, none of these meta-analyses are a reliable basis for forming causation opinions because the vast majority of the studies included in these meta-analyses do not satisfy temporality or look at diagnosed autism or ADHD, as described more fully, *infra*.[16] But even if causal conclusions could be drawn from these meta-analyses, they are assessing a question that is different than the general causation question before the Court because they assess an effect in a non-U.S. population, which is different than a U.S. population.

Rule 702 is clear: if Plaintiffs' experts try to rely on meta-analyses that combine non- U.S. studies, they must use some reliable methodology to reconcile the fact that those very studies do not show a statistically significant association in U.S. populations. *See In re Incretin-Based Therapies Prods. Liab. Litig.*, 524 F. Supp. 3d 1007, 1040 (S.D. Cal. 2021) (excluding expert opinion where expert didn't know if the cited studies were "comparable or not"). Here, Plaintiffs' experts do not do so and their extrapolation from non- U.S. data does not satisfy Rule 702.

* * *

[15] The Ding et al. (2023) meta-analysis is not an anomaly. Indeed, Drs. Ritz, Hu, Gardener, Aschner, and Shapiro rely on multiple published systematic reviews and meta-analyses of epidemiological studies evaluating lead and arsenic levels in children with autism that do not demonstrate associations between heavy metal levels and autism in North America. *See, e.g.*, Ex. 65, at 8-9 (Table 3), 10 (Figure 3) (Shiani, et al., *A systematic literature review on the association between exposures to toxic elements and an autism spectrum disorder* (2023) (reporting statistically significant increased lead in hair of autism cases in eight studies in Egypt, Iraq, Poland, Jordan, Kuwait, Korea, and India, but reporting no statistically significant difference or *decreased* lead levels in autism cases in each of the six studies conducted in the U.S.); Ex. 43, at 311:20-313:24 (Ritz Landon Tr. ) (stating that in the Nakhaee et al. (2022) meta-analysis, of the three studies conducted in North America, two found no association between blood lead level and autism, and one found *lower* hair lead levels in children with autism).

[16] Additionally, all the meta-analyses on which Plaintiffs' experts rely have very high statistical heterogeneity (meaning that the results of the underlying studies are more different than one would expect by chance). As Dr. Ritz admits, high heterogeneity is anything above 50% and high heterogeneity "argues against combining the studies" in a meta-analysis in the first place. Ex. 52, at 261:22-262:1, 262:19-263:2 (Ritz N.C. Tr.); *see also* Ref. Manual at 608 (noting that high heterogeneity "make[s] it harder to trust a single estimate of effect"). Yet, every single meta-analysis on which Plaintiffs' experts rely has heterogeneity substantially above 50% and, indeed, for the vast majority of studies, heterogeneity is above 90%. This is yet another reason why Plaintiffs' experts' extrapolation from these meta-analyses is unreliable.

The impact of any one of these unsupported extrapolations is sufficient to raise a "red flag" in the Court's analysis under Rule 702. *See Downs*, 126 F. Supp. 2d at 1125; *see also Lust by & Through Lust v. Merrell Dow Pharms.*, 89 F.3d 594, 597-98 (9th Cir. 1996) (affirming exclusion of expert based on extrapolation to different endpoint). But the presence of at least *five* points of extrapolation with *no* reliable methodology to explain why such extrapolations are reasonable is altogether fatal to Plaintiffs' experts' opinions. The Court should exclude their "guesswork" as impermissible under Rule 702. *Rosen*, 78 F.3d at 319.

### D. Even If Plaintiffs' Experts Could Extrapolate from Non-Food Studies of Different Endpoints, Windows of Exposure, and Populations, the Data on Which They Rely Do Not Satisfy Basic Scientific Principles Required to Establish Causation.

Even if Plaintiffs' experts could reliably extrapolate from the body of literature examining heavy metal exposures from any source, in any population, during any developmental window (which they cannot), Plaintiffs' experts fail to apply a scientifically sound methodology to their analysis of the literature on which they rely. In analyzing their chosen body of literature, Plaintiffs' experts uniformly minimize—and at times flatly disregard—core epidemiologic principles, downplay results that show *no* association, and rely instead on flawed, cherry-picked studies that they claim support their causation opinions. Among many other things, Plaintiffs' experts claim that numerous studies that fail to establish temporality (i.e., that heavy-metal exposure preceded the onset of autism/ADHD or the outcomes measured in the studies) support their causation opinions—notwithstanding the experts' acknowledgment that temporality is the one bedrock requirement that *must* be met in order to find causation. And when it comes to the (exceedingly small) number of studies that potentially could satisfy temporality, and looked at diagnosed autism and ADHD, Plaintiffs' experts disregard the fact that nearly all of those studies fail to show *any* statistically significant association between heavy metal exposure and autism/ADHD. Plaintiffs' experts' rejection of widely accepted scientific principles in favor of an outcome-driven analysis further underscores why their opinions must be excluded.

### 1. Plaintiffs' Experts Opinions Rely Heavily on Studies That Do Not Satisfy Temporality, a Prerequisite to Reliably Conclude Causation.

It is a bedrock epidemiological principle that to establish causation, there must be reliable evidence that the exposure in question (here, baby food or at least heavy metals) *preceded* the outcome (here, autism/ADHD). This requirement, known as "temporality," is the one component of the Bradford Hill analysis—which several of Plaintiffs' experts purport to apply to reach their causation opinions—that everyone agrees *must* be met to conclude that there is causation. Ref. Manual, *supra*, at 601 ("A temporal, or chronological, relationship must exist for causation to exist."); *see also In re Mirena IUS Levonorgestrel-Related Prods. Liab. Litig.*, 341 F. Supp. 3d 213, 242–43 (S.D.N.Y. 2018) ("[a] cause must precede its effect."), *aff'd sub nom.*, *Coning v. Bayer Pharma AG (In re Mirena IUS Levonorgestrel-Related Prods. Liab. Litig.)*, 982 F.3d 113 (2d Cir. 2020). Yet despite acknowledging this foundational requirement, Plaintiffs' causation experts base their opinions almost entirely on studies that do *not* satisfy temporality. These experts have no coherent explanation for this departure from their own chosen methodology, which renders their opinions unreliable and inadmissible under Rule 702.

Dr. Ritz acknowledges that temporality "is the only component that is necessary for making a causal contribution," which she agreed meant that "temporality is a required component of determining causality." Ex. 1, at 14 (Ritz Rep.); Ex. 26, at 56:6-17 (Ritz MDL Tr.). Drs. Guilarte, Hu, Aschner, and Shapiro acknowledged the same. *See* Ex. 35, at 63:18-20, 114:11-13 (Guilarte MDL Tr.) (stating temporality is "a required component of determining causality" and is a "basic tenet of epidemiology"); Ex. 10, at 235:4-15 (Aschner MDL Tr.); Ex. 51, at 129:24-130:23 (Shapiro N.C. Tr., Vol. I); Ex. 29, at 259:15-260:3 (Hu MDL Tr.).

Plaintiffs' experts also agree that concerns about temporality are particularly acute in the context of studies on heavy metals and autism because there are certain behaviors common in autistic children—such as pica and restrictive eating—that can *increase* their exposure to and absorption of heavy metals, and thereby raise the possibility of reverse causation. *See* Ex. 35, at 156:4-7, 158:1-9 (Guilarte MDL Tr.); Ex. 26, at 280:25-282:2 (Ritz MDL Tr.). Dr. Guilarte, for example, agreed that "pica and prolonged hand-to-mouth behaviors may be one reason why kids

with ASD have elevated levels of heavy metals," and therefore that it is "important to control for pica as a potential confounder to help control for reverse causality." *See* Ex. 35, 156:4-7, 158:1-9 (Guilarte MDL Tr.). Dr. Ritz also agreed that pica may be one reason children with autism have elevated levels of heavy metals. *See* Ex. 26, at 280:25-282:2 (Ritz MDL Tr.). Restrictive eating behaviors among children with autism can also lead to nutrient deficiencies that, in turn, may lead to increased absorption of heavy metals.[17,18] Dr. Guilarte testified that nutrient deficiencies in children with autism may lead to higher lead and arsenic levels. *See* Ex. 35, at 190:22-192:8 (Guilarte MDL Tr.) (agreeing that children with more severe autism symptoms may have more restrictive eating behaviors resulting in deficiencies in important nutrients, leading to higher lead and arsenic levels in various biomarkers). And he agreed that epidemiological studies of heavy metals and autism should control for pica and essential nutrients as potential confounders to address the possibility of reverse causality. *Id.* at 158:1-9; 205:1-7, 208:10-20.

Plaintiffs' experts nonetheless rely on a vast number of heavy-metal studies that cannot establish the required temporal sequence between exposure and outcome—indeed, more than 50 studies cited by Plaintiffs do not satisfy the basic tenet of temporality. Most studies on which Plaintiffs' experts rely are cross-sectional studies or case-control studies, which could not satisfy temporality. Dr. Guilarte, for example, agreed that most of the epidemiological studies in his report could not establish temporality, "meaning that exposure preceded the outcome." Ex. 35, at 111:23-112:2 (Guilarte MDL Tr.). Dr. Hu similarly acknowledged that the majority of the studies cited in his report are meta-analyses of case-control studies, which "typically measure exposure after a child

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[17] Ex. 69 (Baraskewich, et al., *Feeding and eating problems in children and adolescents with autism a scoping review* (2021)); Ex. 82 (Schreck, et al., *A comparison of eating behaviors between children with and without Autism* (2004)). In a recent comprehensive investigation of eating problems, including 103 children with autism, 45.6% were classified as showing substantial food selectivity that was associated with disturbed meal behavior and a general lack of compliance. Ex. 86 (Wenzell, et al., *Clinical Correlates and Prevalence of Food Selectivity in Children with Autism Spectrum Disorder* (2024)).

[18] Ex. 73 (Kinnell, et al., *Pica as a feature of autism* (1985)); Ex. 84 (Kerwin, et al., *Parental report of eating problems and gastrointestinal symptoms in children with pervasive developmental disorders* (2005)); Ex. 74 (Mayes & Zickgraf, *Atypical eating behaviors in children and adolescents with autism, ADHD, other disorders, and typical development* (2019)).

already has autism." *See* Ex. 29, at 260:4-13 (Hu MDL Tr.). Dr. Aschner, for his part, stated that an assessment of temporality was "not part of [his] systemic approach" to studies cited in his report. *See* Ex. 34, at 235:22-236:3 (Aschner MDL Tr.).

Moreover, despite acknowledging the concerns about restrictive eating and reverse causality that apply specifically to studies of heavy metals and autism, Plaintiffs' experts did not attempt to evaluate the impact of nutrient deficiencies in children with autism on levels of lead or arsenic in biomarkers. To the contrary, they acknowledged that it would not even be possible for them to assess this recognized potential confounder based on the literature and data available to them. *See* Ex. 34, at 280:4-21 (Aschner MDL Tr.) ("So as I indicate in my report, because of the competitive nature of these metals, it is certainly possible that the lead levels are increased. There are some deficiencies. I did not analyze the data that are in those papers because I don't have the raw data. So even if I wanted to do it, it would be impossible."); Ex. 26, at 279:4-280:17 (Ritz MDL Tr.) ("Q. Are restricted eating patterns and essential nutrient deficiencies in children with ASD a plausible biological mechanism by which children with ASD have higher lead and arsenic biomarker levels than children without ASD? A. That's a very broad question. I wouldn't know how to answer that."). Nonetheless, these experts all claim that studies that fail to establish temporality or address the risk of reverse causation support their causation opinions.

When pressed to justify their departure from a core aspect of the Bradford Hill analysis, Plaintiffs' experts had no coherent response. Ex. 26, at 56:6-60:16 (Ritz MDL Tr.); Ex. 33, at 231:1-233:5, 272:15-276:6 (Gardener MDL Tr.); Ex. 37, at 451:3-457:15, 476:16-483:12 (Shapiro MDL Tr., Vol. II); Ex. 29, at 259:3-19 (Hu MDL Tr.) (testifying as to temporality that "the lack of evidence for one particular consideration doesn't mean that you can't come to a conclusion on causality."). Their willingness to contradict accepted science—and their own purported methodology—to reach made-for-litigation opinions further confirms that their opinions must be excluded under Rule 702. *See, e.g.*, *Nelson v. Matrixx Initiatives,* No. C 09-02904 WHA, 2012 U.S. Dist. LEXIS 118300, at *27-30 (N.D. Cal., Aug. 21, 2012); *Luttrell v. Novartis Pharm. Corp*. 894 F. Supp. 1324, 1338-1339 (E.D. Wa., 2012).

**2. The Few Studies on Which Plaintiffs' Experts Rely That Satisfy Temporality and Look at Diagnosed Autism and ADHD Do Not Reliably Establish Causation.**

Finally, even the (exceedingly few) studies on which Plaintiffs' experts rely that *do* satisfy temporality and look at diagnosed autism and ADHD do not provide reliable scientific support for Plaintiffs' experts' causation opinions—including because the studies (among other things): focus on the wrong developmental window (*i.e.*, prenatal vs. postnatal); do not show a *statistically significant* association between heavy metal exposure and autism/ADHD; and/or suffer from other defects that prevent them from providing reliable evidence of causation. Plaintiffs' experts, once again, skate past these deficiencies and—without providing any sound scientific explanation or methodology justifying their decision to do so—claim that those studies support their opinions.

**a) Arsenic and Autism.**

As to arsenic, there are simply *no* studies evaluating postnatal exposure to arsenic and autism that satisfy temporality. *See* Ex. 35, at 217:1-15 (Guilarte MDL Tr.). In other words, Plaintiffs do not have a single study that (1) examines arsenic exposure during the time window that everyone agrees applies in this case (i.e., postnatal early life, at an age when children consume baby food), and (2) satisfies the bedrock temporality requirement for causation.

But even if one were to consider the *prenatal* arsenic exposure studies on which Plaintiffs' experts (improperly) rely, those studies too do not provide reliable evidence of an association. Out of only three studies on prenatal arsenic exposure that satisfy temporality and look at diagnosed autism (Skogheim et al. (2021), Dou et al. (2024), and Long et al. (2019)), only one (Skogheim et al. (2021)) reports any statistically significant increased risk. However, Skogheim only reports a statistically significant increased risk for the lower (2$^{nd}$ quartile) of arsenic exposure; it reports no statistically significant increased risk for the higher (3$^{rd}$ and 4$^{th}$ quartiles) of arsenic exposure. Indeed, the highest arsenic exposure level in Skogheim is associated with a non-significantly decreased risk.

**b) Lead and Autism.**

As to lead, there are only three studies—Frye et al. (2020), Arora et al. (2017), and Abdullah et al. (2012)—that evaluate postnatal exposure, evaluate participants with a confirmed diagnosis of

autism, and satisfy temporality. *See* Ex. 35, at 229:3-230:1, 244:24-245:10, 254:8-13 (Guilarte MDL Tr.); *see generally* Ex. 72 (Frye, et al., *Early life metal exposure dysregulates cellular bioenergetics in children with regressive autism spectrum disorder* (2020)); Ex. 68 (Arora, et al., *Fetal and postnatal metal dysregulation in autism* (2017)); Ex. 67 (Abdullah, et al., *Heavy Metal in Children's Tooth Enamel: Related to Autism and Disruptive Behaviors?* (2012)). Not one of these studies showed a statistically significant association between lead and autism during the window when children consume baby food.

Plaintiffs' experts make much of the Arora et al. (2017) study, a small study of twins that found an association between higher levels of lead in infants aged *10-20 weeks* and a later diagnosis of autism. But Plaintiffs' experts repeatedly gloss over the key facts that (1) infants do not typically consume baby food between ages 10-20 weeks, and (2) *no* association was shown for any other postnatal time window studied. *See, e.g.*, Ex. 35, at 231:16-233:1 (Guilarte MDL Tr.); Ex. 29, at 264:2-22 (Hu MDL Tr.). Moreover, even the single postnatal association the Arora et al. (2017) authors identified at ages 10-20 weeks was not statistically significant after the authors corrected for multiple comparisons—as Plaintiffs' experts also admit. *See* Ex. 35, at 237:20-238:6 (Guilarte MDL Tr.) ("Q. . . . After correction from multiple comparisons, there were no statistically significant associations at any point in time between lead levels and ASD in the Arora study, correct? A. Based on this information, correct."); *see also* Ex. 43, at 239:21-240:1 (Ritz Landon Tr.); Ex. 68, at 4 (Arora, et al., *Fetal and postnatal metal dysregulation in autism* (2017)).

Even if one were to consider the *prenatal* exposure studies on which Plaintiffs' experts (improperly) rely, those studies also do not provide reliable evidence of an association. Out of only four prenatal studies on lead exposure that satisfy temporality and look at diagnosed autism (Long et al. (2019), Skogheim et al. (2021), Wegmann et al. (2023), and Dou et al. (2024)), *three* reported no statistically significant increased risk of autism from prenatal lead exposure. *See* Ex. 66, at 15-19 (Long, et al., *Autism spectrum disorders, endocrine disrupting compounds, and heavy metals in amniotic fluid: a case-control study* (2019)); Ex. 77, at 5 (Skogheim, et al., *Metal and essential element concentrations during pregnancy and associations with autism spectrum disorder and attention-deficit/ hyperactivity disorder in children* (2021)); Ex. 78, at 11-12 (Wegmann, et al.,

*Identification of potentially relevant metals for the etiology of autism by using a Bayesian multivariate approach for partially censored values* (2023)). The fourth study (Dou et al. (2024)) found no association between maternal urinary levels of lead and offspring autism, but reported a small association (RR 1.23, 95% CI 1.01-1.54) between maternal blood levels of lead and offspring autism. *See* Ex. 71, at 8 (Dou, et al. *Exposure to heavy metals in utero and autism spectrum disorder at age 3: a meta-analysis of two longitudinal cohorts of siblings of children with autism* (2024)). However, this study failed to adjust findings for multiple testing—which Dr. Guilarte agreed should have been done to decrease the likelihood of finding false associations due to chance. *See id.* at 3*; see also* Ex. 35, at 74:10–75:1 (Guilarte MDL Tr.). And in any event, as Plaintiffs' experts all acknowledge, a single study showing a small positive association cannot support a finding of causation. *See, e.g.*, Ex. 52, at 143:24-144:2; 337:14-20 (Ritz N.C. Tr., Vol. I).

**c) Lead and ADHD.**

The only study addressing lead and ADHD that satisfies temporality, evaluates diagnosis of ADHD, and assesses postnatal exposure (Ji 2018) is problematic for other reasons.[19] Ji et al. (2018) found *no* increased risk of ADHD in children with blood lead levels below 5ug/dL during the developmental window when children typically eat baby food. *See* Ex. 91, at 5 (Ji, et al., *A Prospective Birth Cohort Study on Early Childhood Lead Levels and Attention Deficit Hyperactivity Disorder: New Insight on Sex Differences* (2018)); *see also* Ex. 35, 295:7-16 (Guilarte MDL Tr.). As discussed above, the average blood lead level of U.S. children is only *0.6* ug/dL. *See* EPA, Biomonitoring—Lead (2023), Biomonitoring - Lead | US EPA. The Ji et al. (2018) study also found *no* statistically significant association between lead exposure and ADHD in female children at any age, did not control for genetics, and did not control for socioeconomics. *See* Ex. 35, 295:13-16 (Guilarte MDL Tr.); *see generally* Ex. 91 (Ji, et al., *A Prospective Birth Cohort Study on Early Childhood Lead Levels and Attention Deficit Hyperactivity Disorder: New Insight on Sex*

[19] None of Plaintiffs' experts opine that exposure to arsenic in baby food is associated with ADHD or that exposure to any other heavy metals (e.g., cadmium and mercury) is associated with either autism or ADHD. Absent supporting expert opinions, those claims fail as a matter of any state's law.

*Differences* (2018)). As Dr. Guilarte acknowledged, there is a known association between low socioeconomic status and higher exposure to heavy metals, and "it would be important" to control for socioeconomic status in epidemiological studies of heavy metals and neurodevelopmental outcomes. Ex. 35, 103:3-20 (Guilarte MDL Tr.).

Even if one were to consider the prenatal exposure studies on which Plaintiffs' experts (improperly) rely, those studies also do not provide reliable evidence of an association. In fact, only one study satisfied temporality and looked at prenatal lead exposure and diagnosed ADHD. That study (Skogheim et al. (2021)) reported no statistically significant increased risks for lead exposure at any level and ADHD. Ex. 77, at 11 (Skogheim, et al., *Metal and essential element concentrations during pregnancy and associations with autism spectrum disorder and attention-deficit/ hyperactivity disorder in children* (2021)).

Finally, to justify their reliance on many of the studies that they claim support a causation opinion, Plaintiffs' experts disregarded widely accepted principles on the importance of statistical significance. They agree that statistical significance is the generally accepted methodology for determining the likelihood that a study's findings are due to chance versus a reliable, replicable association. *See* Ex. 35, at 72:6-10 (Guilarte MDL Tr.); Ex. 43, at 117:4-14, 136:8-12 (Ritz Landon R. Tr.); Ex. 29, at 90:15-92:9 (Hu MDL Tr.). Indeed, Dr. Guilarte testified that results that are not statistically significant are interpreted as showing "no association" and that this is "just a generally accepted and reliable statistical methodology." Ex. 35, at 72:15-19, 73:16-74:2 (Guilarte MDL Tr). Yet, Plaintiffs' experts' causation opinions rest almost entirely on studies that show *no* statistically significant association. The experts' decision to rely on non-statistically significant findings as evidence of causation only further demonstrates their unreliable and not generally accepted methodology. Under the Federal Rules, district courts have a "function as a gatekeeper; it is not for the courts to be the pioneers, forging new trails in scientific thinking, especially when that means departing from well-established research principles, such as the principle of statistical significance." *In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig.*, 26 F. Supp. 3d 449, 456 (E.D. Pa. 2014).

Indeed, Plaintiffs' experts' reliance on non-statistically significant findings is particularly egregious because they cherry-pick non-statistically significant increased risks, while ignoring non-

statistically significantly (or even statistically significantly) decreased risks, in a result-driven manner. *See e.g.*, Ex. 53 at 389:6-17 (Ritz N.C. Tr., Vol. II); Ex. 52 at 141:24-142:7 (Ritz N.C. Tr., Vol. I). "Result-driven analysis, or cherry-picking, undermines principles of the scientific method and is a quintessential example of applying methodologies (valid or otherwise) in an unreliable fashion." *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prods. Liab. Litig. (No II)*, 892 F.3d 624, 634 (4th Cir. 2018). "An expert must not cherry-pick from the 'scientific landscape and present the Court with what he believes the final picture looks like.'" *Daniels-Feasel v. Forest Pharms., Inc.*, No. 17 CV 4188-LTS-JLC, 2021 WL 4037820, at *5 (S.D.N.Y. Sept. 3, 2021) (quoting *In re Rezulin Prod. Liab. Litig.*, 309 F. Supp. 2d 531, 563 (S.D.N.Y. 2004)). "Sound scientific methodology in assessing general causation requires an expert to evaluate 'all of the scientific evidence when making causation determinations.'" *Id.* at *5 (quoting *In re Zoloft (Sertraline Hydrocloride) Prod. Liab. Litig.*, 26 F. Supp. 3d 449, 463 (E.D. Pa. 2014)); *see also In re Incretin-Based Therapies Prods. Liab. Litig.*, 524 F. Supp. 3d 1007, 1035-36 (S.D. Cal. 2021) (excluding expert opinion because expert did not update his prior analysis to address changed landscape for incretins and did not "consider[] all available clinical data and peer-reviewed literature relating thereto[,]" so his data was "misleading" and "unreliable"); *In re Viagra (Sildenafil Citrate) & Cialis (Tadalafil) Prods. Liab. Litig.*, 424 F. Supp. 3d 781, 797 (N.D. Cal. 2020) (excluding expert opinion of epidemiologist because the "application of the Bradford Hill factors appears result-driven"). Plaintiffs' experts utterly failed to do so here.

## IV. CONCLUSION

For the foregoing reasons, Defendants respectfully request that the Court exclude the general causation opinions of Drs. Aschner, Gardener, Guilarte, Hu, Ritz, and Shapiro.

Dated: September 26, 2025

DLA PIPER LLP (US)

By: /s/ *Brooke Killian Kim*
Brooke Killian Kim
Mary Gately (*pro hac vice*)
Loren H. Brown (*pro hac vice*)

*Attorneys for Defendant Nurture, LLC*

Dated: September 26, 2025

*WILLIAMS & CONNOLLY LLP*

*By: /s/ Neelum J. Wadhwani*
*Neelum J. Wadhwani (SBN 247948)*
*Joseph G. Petrosinelli (pro hac vice)*

*WHITE & CASE LLP*

*By: /s/ Bryan A. Merryman*
*Bryan A. Merryman*

*Attorneys for Defendant Gerber Products Company*

Dated: September 26, 2025

KING & SPALDING LLP

By: */s/ Livia M. Kiser*
Livia M. Kiser
Michael Anthony Lombardo
Todd P. Davis (*pro hac vice*)

*Attorneys for Defendants Beech-Nut Nutrition Company and Walmart Inc.*

Dated: September 26, 2025

COVINGTON & BURLING LLP

By: /s/ *Michael X. Imbroscio*
Michael X. Imbroscio (*pro hac vice*)
Phyllis A. Jones (*pro hac vice*)
David N. Sneed (*pro hac vice*)
Elizabeth T. Fouhey (*pro hac vice*)

*Attorneys for Defendant The Hain Celestial Group, Inc.*

Dated: September 26, 2025

GORDON REES SCULLY MANSUKHANI, LLP

By: /s/ *Michael R. Klatt*
Michael R. Klatt (*pro hac vice*)
Nancy Mae Erfle (*pro hac vice*)

*Attorneys for Defendant Sprout Foods, Inc.*

Dated: September 26, 2025

DECHERT LLP

By: /s/ *Hope Freiwald*
Hope S. Freiwald (*pro hac vice*)
Michelle H. Yeary (*pro hac vice*)
2929 Arch Street

**CERTIFICATE OF SERVICE**

I certify that on September 26, 2025, I electronically filed the foregoing **DEFENDANTS' MOTION TO EXCLUDE PLAINTIFFS' CAUSATION/ EPIDEMIOLOGY EXPERTS (BRIEF 3)** using the ECF system, which sent notification of such filing to all counsel of record.

*/s/ Brooke K. Kim*
Brooke K. Kim